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TO THE

FORMER AND PRESENT HOUSE-PHYSICIANS AND CLINICAL CLERKS
OF KING'S COLLEGE HOSPITAL,

WHO HAVE FOR NEARLY TWENTY YEARS KINDLY AND

ABLY ASSISTED THE AUTHOR

IN HIS CLINICAL OBSERVATIONS,

These Pages are Dedicated,

WITH EVERY FEELING OF AFFECTION AND THANKFULNESS,

BY THEIR SINCERE FRIEND,

THE AUTHOR.

December 28, 1859.

# PREFACE.

THE design of the lectures published in the present volume, is to describe and illustrate by examples the clinical history and treatment of the more important acute diseases.

There will (the author believes) be found in the following pages evidence enough to show that the ordinary so-called antiphlogistic treatment is unnecessary (to say the least) for the cure of acute internal inflammations; and that the supposed necessity for such treatment rested upon an untenable hypothesis respecting the nature of inflammation and of fevers, and cannot be regarded as a legitimate induction from accurately observed clinical facts.

The conclusions, which the clinical observations detailed in the lectures tend more or less to establish, may be summed up in the following propositions:—

1. That the notion so long prevalent in the schools, that acute disease can be prevented or cured by means which depress and reduce vital and nervous power, is altogether fallacious.

2. That acute disease is not curable by the direct influence of any form of drug or any known remedial agent, excepting when it is capable of acting as an antidote, or of neutralizing a poison, on the presence of which in the system the disease may depend (*materies morbi*).

3. That disease is cured by natural processes, to promote which, in their full vigor, vital power must be upheld. Remedies, whether in the shape of drugs, which exercise a special physiological influence on the system, or in whatever form, are useful only so far as they may excite, assist, or promote these natural curative processes.

4. That it should be the aim of the physician (after he has sedulously studied the clinical history of disease, and made himself master of its diagnosis), to inquire minutely into the intimate nature of these curative processes—their physiology, so to speak; to discover the best means of assisting them, to search for antidotes to morbid poisons, and to ascertain the best and most convenient methods of upholding vital power.

If one may venture a suggestion respecting the future of pathology, and of practice founded on it, it would be that a time is not far distant when all men who practise medicine in a scientific spirit, and divested of the trammels of routine, will discard the distinction of acute inflammations and acute disease in general, into *asthenic* and *sthenic*—that all these maladies will be regarded as more or less asthenic, and as promoting more or less an undue waste of tissue, and that, in treatment, an object of primary importance will be the early adoption of means to uphold vital power, and the watchful and continued use of them throughout the duration of the case.

It will not be affirmed by any one that the doctrines of a science so abstruse and so difficult as pathology, should not be reviewed and reconsidered from time to time. There never was a period when a candid and ample reconsideration of general pathology promised more fruitful results than the present. Our vastly extended acquaintance with anatomy and physiology, the greatly enlarged security of the basis on which our knowledge of function rests, the much increased accumulation of facts of clinical history, all afford most important data for new inductions. And I would remark that such inductions ought to be made from the deranged functions of the living rather from the facts of morbid anatomy, which properly should rank with the facts of clinical history, and which, in reality, are inferior in value to most of the phenomena of disease during life, being no more than marks of the ravages of disease, and affording comparatively little insight into its intimate nature. The real basis of all pathological inquiry must be clinical research, made with the fullest appreciation of the facts of anatomy and physiology; *mere* morbid anatomy leads necessarily to erroneous views of pathology and practice.

Such a review of pathological doctrine, as I have alluded to, will assign its true value to the influence of the *quantity* of blood in the production of disease; will determine what is the real point of departure of morbid change, whether it is due to a superabundant or a deficient blood supply; or whether the condition of the blood supply is the consequence of a primary morbid change, such as a disturbed innervation, or a contamination, or waste of the elementary tissue.

The following problem lies at the root of the pathology of acute disease, and it has never yet received an adequate explanation, and is uniformly ignored by the zealous advocates of the so-called antiphlogistic method:—

A man has a patch of pneumonia in the base of his left lung, brought on (he conjectures) by some exposure to cold. Why is it in his *left* lung? why at the base rather than the apex? how is it limited to a certain patch? In other words, what is the proximate cause of this localized derangement of nutrition?

If so much has yet to be determined as regards the very alphabet of pathology, it cannot surely be thought presumptuous to question the soundness of the practice founded on such crude doctrines.

And more especially are we justified in such a course, when it is considered that much of the practice of former days rests upon the insecure foundation of a partial and imperfect diagnosis of the primary disease, and a very inadequate interpretation of the subsequent phenomena of the case. Thus, in many instances the practitioner found himself treating a disease of the clinical history of which he had but a very imperfect knowledge, and on these occasions, he would be led to attribute changes in the symptoms (whether for better or for worse), which were essentially part of the ordinary course of the malady, to the influence of certain remedies. The temptation to draw hasty conclusions as to cause and effect, and to adopt the *post hoc ergo propter hoc*, so common among all classes, unlearned or learned, often likewise stood in the way of sound reasoning upon these subjects.

Did space permit, it would be easy to adduce many instances to show that a more exact diagnosis must necessarily lead to altered views of practice. A few may be briefly referred to.

The precise discrimination of the different forms of continued fever has arisen out of the clinical investigations of the last fifteen or twenty years. And it is now ascertained that continued fever may be caused by any one of three separate poisons, each of which developes its characteristic phenomena, namely, those of typhus, typhoid, or pythogenic and relapsing fevers. Nevertheless, there is good reason for believing that any two of these poisons may coexist in the same individual and produce their special phenomena. Dr. Murchison's researches render it highly probable that the great epidemics which formerly ravaged Ireland, and some of the large towns of Scotland, were chiefly the fever which is called *relapsing*, and that it was by this form of fever that the practice of bleeding and low diet was best borne.

However this may be, it is clear that even just to the present day physicians were not in a position to discriminate whether certain changes in the phenomena of the disease were due to the influence of their remedies, or were simply part and parcel of the ordinary train of the clinical phenomena of the disease.

The whole class of the so-called *apoplectic* diseases must now be viewed, as regards their pathology and treatment, in a totally different light from that in which they were regarded formerly, even so lately as the celebrated work of Abercrombie. Both the pathology and practice of that able physician must now be, with but little exception, entirely discarded.

And to what is this owing? Undoubtedly to a more extended knowledge of clinical history, and to the consequent more precise discrimination of the different forms of brain disturbance, which lead to comatose and paralytic phenomena. For example, the author has shown long since that many cases of hemiplegia are the result, not of a clot in, or of a rupture of fibres of the brain, but simply of the influence of an epileptic fit. The history of these cases is, that the patients suddenly fall into epileptic coma, with or without convulsion, and that they emerge from it with more or less perfect hemiplegia. This paralysis often gets well with remarkable rapidity after a few hours, very often after a few days, and sometimes after some weeks.

A case of this kind falling into the hands of a practitioner accustomed to use the lancet freely in diseases tending to coma, (the so-called congestion of the brain), would not suffer any disadvantage from a moderate bleeding and from purging; the patient would speedily recover, and the case would be quoted as a glaring instance of the excellent effect of the treatment, whereas an exact diagnosis might have saved unnecessary practice, and a familiar knowledge of clinical l [illegible] ave enabled the practitioner to have foreseen and foretold the course which the disease would be likely to take.

The much vaunted powers of mercury as a remedy, not only to promote the resolution of acute inflammation, but also to cause the absorption of its product, lymph, rests first upon a false analogy; and, secondly, upon imperfect knowledge of clinical history.

It was found that iritis, the result of the influence of syphilis, was cured under the use of mercury with a rapidity and certainty which did not belong to any other kind of treatment. Lymph effused in more or less quantity upon the surface of the iris, and even recent adhesions gluing the margin of the pupil to the capsule of the lens, quickly melted away under the peculiar change which mercury was capable of inducing.

*Primâ facie*, there was no more reasonable suggestion than that mercury would exercise a similar influence on inflammations of like tissues to the membrane of the anterior chamber, and promote the removal of any lymph that might be effused upon them, preventing adhesions or dissolving them if formed.

But although it was a perfectly reasonable suggestion to give full trial to the use of mercury in inflammations of serous membranes, the analogy did not justify the expectation of such decisive results as were obtained in syphilitic iritis, although sufficient to call for experiments. In fact, the analogy was inexact: there was no further resemblance between syphilitic iritis and rheumatic pericarditis or pleurisy, than in the tendency of both inflammations to develope lymph, and to cause adhesion of opposed surfaces. Nor in any of their other effects was there any such marked similarity between the syphilitic and the rheumatic poisons as would fully justify the expectation that the experimental trial of mercury for the cure of such inflammations would prove successful.

And what has been the result of the long-tried use of mercury in both affections, syphilitic as well as rheumatic? Why, that whilst, in the former, mercurial treatment has never ceased to find favor with practical men, in the latter, such has not been the case. No one would now venture to assert that mercurial influence, however quickly induced, ever checked pericarditis or pleurisy; nor would it be easy to adduce an instance in which, with any reasonable degree of certainty, it could be stated that mercury broke down adhesions, or prevented their occurrence.

Examples were no doubt of frequent occurrence in which such effects *appeared* to follow the use of mercury. But a more intimate acquaintance with clinical history has taught physicians that changes are apt to occur which simulate the absorption of a lymph-deposit. It is very common to find a marked pericardial friction sound disappear for a time, and the hopeful practitioner is led to regard this as the result of his remedies, sometimes of a few leeches or a cupping, sometimes of a blister, but more especially of the use of mercury. In a day or two, the friction sound returns, and the practitioner is forced to conclude that his remedies have not produced the desired result. And there are the best reasons for inferring that the early temporary suspension of the friction sound is due in a large number of cases to a slight liquid effusion, which separates the opposed rough surfaces, and so destroys the friction sound, which, however, returns on the reabsorption of the liquid. Moreover, it is now proved by a multitude of examples (and some will be found recorded in the following pages), that pericarditis will do perfectly well without mercury, nay, better than with it; and that in general the real benefit which the patient derives is to be referred to the opium with which the questionable mercury is combined.

How often and often has the author most anxiously watched a mitral bellows-murmur, caused by recent endocarditis in patients under mercurial treatment, hoping to discover that it had disappeared under the mercurial influence! Yet in his whole experience he is unable to discover a single case in which such a murmur had been even modified by any influence save that of good nourishment, as tending to maintain a normal state of blood, and of time, as furnishing opportunity for the mechanical wearing down (by attrition) of the deposited lymph.

A curious instance may be mentioned in illustration of the way in which an erroneous opinion may be readily formed with respect to the effects of remedies. The author attended, along with a medical friend, a tradesman of middle age, who became quickly, although not suddenly, comatose, with hemiplegia of the right side, and marked rigidity of the muscles of the arm and leg. It was difficult to determine whether the symptoms indicated a superficial apoplectic clot, or were due to a patch of inflamed brain in the left hemisphere. It was resolved to act on the latter view, and mercury was given freely with the intention of producing salivation. About the second or third day of this treatment the patient recovered his consciousness, although retaining a certain degree of somnolency; the paralyzed limbs regained a greater degree of power and the muscles became less rigid. During this and the succeeding day everything favored the conclusion that the mercury was telling upon the inflammatory process, and that it was undergoing resolution. The gums became affected just at this time. But, in another day, the hopes of the patient's friends and attendants were seriously checked by the rapid recurrence of the comatose and paralytic symptoms, in a more severe form, leading to a profound coma, under which the patient succumbed.

The inspection after death showed a considerable apoplectic clot on the surface of the left hemisphere, causing a deep indentation on the convolutions, which did not disappear when the clot was removed, such was the degree of pressure on the cerebral surface. On cutting through the clot, it was found to consist very distinctly of two portions, one brownish in color, and looking old; the other consisting of dark, currant-jelly-like coagulum, which had been only quite recently effused.

It was plain, then, in this case, that the original cause of the symptoms was a meningeal effusion of blood compressing the brain to a very great extent. After a few days, the watery part of the coagulum was absorbed, and the shock which the brain had received at the first effusion of blood had subsided. It was then that the restoration of consciousness and the improvement in the other symptoms, which were attributed to the influence of mercury, took place. This improvement, however, soon gave way before a fresh hemorrhage, which led quickly to a fatal result.

Had an exact diagnosis been possible with certainty in this case, the patient would have been spared the mercurial course to which he was subjected, and the physicians would have had nothing else to do but to support the powers of life, with the hope, that by the brain adapting itself to the pressure, and the gradual absorption or contraction of the clot, the life of the patient might be considerably prolonged. And, indeed, this would most probably have been the case in this patient, had it not been for the second hemorrhage.

Enough has been said, the author hopes, by way of apology, for his venturing to dissent from current views of pathology and practice, sanctioned as they are by great names, both dead and living—for which the author will yield to no one in admiration and respect; but

Amicus Plato, amicus Socrates, magis amica veritas.

The author has not referred to the hypothesis suggested by some who admit the necessity of a considerable modification of practice in the treatment of acute diseases; namely, that the type of disease has undergone material change of late years, and has assumed a much lower grade as regards vital power, owing either to some change in the human constitution, or to some atmospheric modification which has taken place in recent times. It is supposed that this modification dates from the period of the first introduction of cholera into these countries.

Upon this point, the author can say that he has been a not careless observer of disease for several years antecedent to the first cholera epidemic. At no time was the antiphlogistic treatment (so called) more rife than some years prior to the cholera epidemic, and many excellent observers were then beginning to see that it was carried too far, and was inadequate for its object in either cutting short or curing disease. Certainly opportunities of studying the morbid anatomy of acute diseases, pneumonia, pericarditis, endocarditis, pleurisy, were then, and for many years afterwards, much more common than now, when such inspections are among the least frequent in our hospital theatres. The author has notes of many cases treated in this way, which he is confident would have recovered, had vital power been not only spared but upheld.

The author would venture to doubt the proposition that disease is of a lower type now than it was twenty or thirty years ago. Certainly we have long been spared those ravaging epidemics of fevers, dysenteries, exanthemata, all of which exhibited innumerable examples of the lowest type of disease. Their comparative disappearance now is due in part, no doubt, to the improved condition of the people, better food, better clothing, cleaner and better ventilated dwellings, and to many wise preventive sanitary measures. But, on the other hand, population is vastly increased, overcrowding exists to a large extent, and were disease of a very low type, it would spread freely, and epidemics would be common. It is well known that such is not the case, and that the fevers which were formerly the scourge of the poor, occur now on a very limited scale.

In concluding these remarks, which have extended further than the author intended, it only remains for him to return his most cordial thanks to his highly intelligent friend and former pupil, Dr. Liveing, for his invaluable aid in carrying these pages through the press, and to his friends, Dr. Hyde Salter, Professor Beale, and Dr. Conway Evans, by whom several of the lectures were first reported.

26 Brook Street, Grosvenor Square,
December 28, 1859.

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# CLINICAL LECTURES

ON VARIOUS

# ACUTE DISEASES.

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## LECTURE I.

### *On Rheumatic Fever.*[1]

Gentlemen,—The appropriate treatment of Rheumatic Fever is still, in some degree, a *vexata quæstio.* I propose, therefore, in this and one or two other lectures, to call your attention more especially to this subject; and, by way of introduction, I shall make some remarks on the clinical history and pathology of this interesting form of acute disease.

Case I. (Vol. xxiii, p. 184.) The case by reference to which I shall particularly illustrate my observations is that of Elizabeth Stocking, aged 23 years, admitted on the 19th of April, 1848, and still in the hospital,—a case in which the prominent characteristics of the disease are very well marked, and which, therefore, may be properly selected as a good example of the malady.

Let me take this opportunity of recommending you to study with care, by taking full and daily notes of them, a few cases of this disease. It is a disease which, by and by, you will be frequently called upon to treat; we are seldom without several examples of the disease in the hospital; and, by taking careful notes of some eight or ten of these cases now, you will so impress upon your minds the history and symptoms of the disease, that you will be well prepared to treat them for yourselves, and each new case will be the more profitable to your practical knowledge. This is the more to be recommended, because rheumatic fever exhibits remarkably little variety of symptoms, or difference of phase. In one case the symptoms may be more severe than in another; but the same essentials which characterize the disease are present in all. Therefore, I say, study a few cases carefully, and you will get a good knowledge of this disease before you are called upon to treat it on your own responsibility.

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[1] This lecture was delivered at King's College Hospital in 1848.

The case of Elizabeth Stocking affords, as I have said, a good opportunity of studying the characteristic symptoms of rheumatic fever. The two most prominent features are: First, a special fever, of the continued kind, varying in intensity in the different cases, but always maintaining the same essential characters. This fever is the essence of the malady—the nucleus, as it were, around which all the other symptoms are grouped. Secondly, a peculiar affection of the joints, involving more or less swelling of them, and also pain, which is aggravated by motion.

The fever may exist without the affection of the joints, and it may be accompanied even by an internal inflammation, such as pleurisy, or pericarditis, or endocarditis, as I have witnessed in several examples. But the articular affection never exists without the fever. You will, therefore, not regard the fever as merely symptomatic of a peculiar morbid state of joints; it is, in truth, a fever *sui generis*, of which the articular affection and the other phenomena are but clinical features—attendant symptoms, which may or may not occupy a prominent position. But as the articular affection is very commonly present, and must necessarily demand much of your attention, since it gives rise to much of the patient's suffering, I will assign the first place to the few remarks I have to offer respecting it.

The articular affection almost always commences in the lower joints, and then travels up to the higher; thus it is first found in the ankles and knees, and then it goes to the elbows and wrists. The hip escapes more frequently than any large joint; the shoulder is much more commonly implicated than the hip. When the hip is severely attacked the patient suffers much; the other joints have the affection comparatively mildly; and in several instances it has seemed to me as if the whole force of the rheumatic inflammation had spent itself upon one hip joint.

The implication of the joints is almost always shown by what may be considered its peculiar characteristic—swelling. Almost invariably there is an increase of the synovial secretion, sometimes to a very great extent, so as to prove a source of great annoyance to the patient. The synovial membranes in this condition are highly vascular; so much so, that I have sometimes seen them, in cases where I have had an opportunity of examining them, as red as the conjunctiva when in a state of violent inflammation.

Another characteristic of the disease is its tendency to shift its position. To-day it will be in the *right* knee, which will be swollen, hot, and tender; to-morrow all this will have disappeared, and you will have the same symptoms in the *left*. This erratic tendency—this disposition to wander from joint to joint—is a symptom which you should carefully keep in mind; where it exists in a very marked degree it must be considered a bad feature, indicative of a low form of the disease, and a low state of the vital powers; and it is to cases in which this symptom is prominent that depressing treatment is found to be particularly prejudicial, often aggravating the disease generally, and this feature of it in particular.

This erratic tendency is present, not only in rheumatic fever, but likewise in the analogous disease of gout. It was this disposition to shift from one place to another that led the old writers to regard the internal inflammations, which are apt to come on in the course of these diseases, as "metastatic"—an idea which, however it may have some degree of support in gout, is inadmissible in rheumatic fever. It by no means follows that an inflammation of an internal part should be a metastasis of an external inflammation, even should the latter diminish or cease on the appearance of the former. A strong objection to the doctrine of metastasis is founded on the fact that internal and external inflammations often manifest themselves simultaneously, and very frequently the internal inflammation comes first. Moreover, it rarely happens that the external inflammation becomes diminished or exacerbated by the increase or diminution of the internal, and the converse.

Another feature of this disease is the profuse sweating by which it is accompanied. This is a special phenomenon of the fever. It is not distinctly of a critical or sanitary nature, as we sometimes see it in other fevers; for the sweats do not produce any marked immediate good effect, either on the joints which are implicated in the disease, or on the general state of the patient. In Stocking's case the sweating was profuse: you doubtless recollect how it poured forth from the patient's head and chest, and, indeed, from the surface of her body universally; and from that you may judge how much fluid must have escaped through the channel of the sudatory apparatus. I must say, however, that I do not regard these sweats as otherwise than salutary within certain limits; I think that, in the early days of the fever, they should be encouraged as an important medium for the elimination of noxious matter from the system, and that you ought to be cautious how you stop such sweats, except where they are distinctly debilitating to the patient. Large quantities of free acid are carried off by these sweats: you remember that, on several occasions, we applied litmus to the skin of this patient, and that it always was deeply reddened. In contrast with this extraordinary action of the skin, we remark generally, as it was with our patient Stocking, a deficiency in the quantity of the urine, and an abnormal condition of it; that fluid being loaded with lithates and purpurates, and even oxalates, and sometimes, as in a patient now in the hospital, containing blood: the kidneys are in some degree irritated; less water passes off by them, but apparently a large amount of solid ingredients.

Another symptom, which always accompanies this disease more or less, is a peculiar furred condition of the tongue. This is very characteristic, and will be readily recognized by an experienced eye as distinctive of the rheumatic fever. A thick fur covers the tongue like a wet blanket, and it is not until the fever gives way that this fur begins to pass off. The state of the tongue is the best index to the true condition of the patient; so long as it continues furred, you cannot say that you have succeeded in thoroughly eradicating the rheumatic state; and I would warn you not to be confident in the result of your treatment, unless you see the tongue become quite clean. Even although the pain in the joints and other external signs may have been subdued, yet, if the tongue remain furred, I should greatly fear that the patient may suffer a relapse, or that he may linger on in the rheumatic state for a considerable time.

Further, we had in our case an [illegible] n which rheumatic inflammation affects the heart. On May the 25th, about the thirteenth day of the disease, a rubbing sound was heard over the base of that organ, leading to the conclusion that there had been an effusion of lymph on the opposed surfaces of the pericardium. This was evidently not metastatic—in other words, there was no direct transference of the inflammation from the external parts to the heart—because it coexisted with an undiminished, or but slightly diminished, inflammation of the joints. All these symptoms—namely, the articular swellings, the profuse sweats, the high-colored and loaded urine, the furred tongue, the tendency to heart affection—are present in all cases of rheumatic fever; nor can we regard a case as of this nature in which these symptoms do not exist. In too many the heart affection actually takes place; sometimes it ushers in the attack, and takes precedence of the articular affection; in all it is to be apprehended, and, if possible, guarded against.

There are, however, two points in the case before us which are peculiar, or, at least, which do not occur constantly in cases of rheumatic fever. First, you will remember that, at several of our visits, I pointed out to you on the skin of this patient a copious eruption of what have been called *sudamina* or *miliary vesicles:* they were scattered all over the surface of the thorax, and if you passed your finger over the skin, you found it rough. These sudamina are seen, on close examination, to be minute vesicles, filled with pellucid fluid. They do not especially belong to rheumatic fever, but they are characteristic of a sweating state. If a patient, suffering under typhus fever, pneumonia, phthisis, &c., sweats profusely, these sudamina are apt to appear upon the skin in great numbers. The presence of the vesicles must not be regarded as indicative of any special form of disease, but merely as an accompaniment of a state of very free perspiration.

The second peculiarity in the case of this woman is, that the rheumatic fever followed quickly upon the puerperal state. The connection between rheumatic fever and deranged uterine secretions is very remarkable. Some of the most severe cases I have ever seen have followed dysmenorrhœa. It would seem as if, in these cases, the uterus were but imperfectly evacuated, and its contents becoming decomposed, and getting into the circulation, produced a morbid state of the blood, which gives rise to the symptoms under which the patient labors, and requires for its cure the elimination of the unhealthy material by the various emunctories—a state similar and analogous to pyæmia.

Not unfrequently, after the puerperal state, the patient exhibits all the symptoms of ordinary rheumatic fever: the same profuse sweats, the swollen joints, the fever, the lithic urine. But in some cases the disease runs a more formidable course; the joints, instead of getting better after a time, continue to get worse, till at last the cartilages ulcerate, pus is secreted in large quantities, and fills the synovial membranes to distention; the articular extremities of the bones are laid bare, and the rough osseous surfaces grate against each other when the limb is moved. I have seen all the large joints in this condition. At the same time deposits of pus form in the muscles, and in other parts, even in the eyes. Some of the French writers describe this disease under the name of "*puerperal acute rheumatism.*" It is, in fact, a form of puerperal fever, due to inflammation of some of the uterine veins; this gives rise to the formation of pus, which, infecting the blood, excites articular and other inflammations in its passage through the circulation. Such cases throw light on the pathology of rheumatic fever, and show how a morbid matter, generated at one part of the circulation, and carried throughout it, may occasion serious disturbance in the local nutrition of the various parts through which it may be undergoing elimination, and give rise to a train of symptoms, closely resembling, and not to be distinguished (save by the history) from those of rheumatic fever.

In our patient Stocking there was some morbid state of the uterus prior to the development of the rheumatic condition. Immediately after her confinement she seems to have had symptoms of peritonitis, which appeared to yield to treatment; but she had not long recovered from these symptoms when the rheumatic condition showed itself.

I think it will be as well, before proceeding to the treatment, to adduce some other cases for the further illustration of the preceding remarks as well as for after reference.

CASE II. (Vol. xxxvi, p. 227.) The second case is th [illegible]
Green, a girl in her 16th year, who ca
hospital in January, 1853. This c

more severe form of cardiac complication, and is also instructive as regards the treatment. Five days before her admission, on the 8th of January, she was taken ill with pain and swelling of both knees; she was therefore placed under medical treatment, and, among other things, took gray-powder *until slight ptyalism was produced.* While in this condition, *with her gums still sore,* during the night immediately preceding her admission to the hospital, she suffered from uneasiness about the chest with some cough; there was also a considerable discharge of blood from her throat or nose, to which she has been subject.

On admission, she was suffering from pain in both knees, both shoulders, and ankles; her pulse numbered 110, and respirations 36 in a minute; a slight sound, like a bellows'-sound, accompanied the systole, and was heard best over the base of the heart. A mustard poultice was applied to the chest, and followed by a blister. She commenced taking one grain of opium every three hours, and large sweating doses (six drachms) of the liquor ammoniæ acetatis with camphor mixture.

The next day, on examining her chest, I detected a loud, harsh, to and fro, pericardial rubbing sound; this was audible all over the region of the heart. On the 10th we have the following note: "She complains of much pain in her elbows, knees, and ankles; pulse 106, full and strong; tongue very red where not coated with a whitish-yellow fur; she sweats profusely, sleeps little, and looks pale and anæmic. Her pupils are but slightly affected by the opium." The blister was then dressed with unguentum sabinæ. On the 11th, the pains in the joints, especially in the left knee and right elbow, were aggravated; the sweating had abated, and her skin was hot and dry; the tongue dry and red. The to and fro rubbing sound was very loud and harsh, and heard very distinctly in the course of the left subclavian artery and underneath the left clavicle,—as if the pleura over the pericardium were involved; the rubbing, however, was not synchronous with respiration. A blister was again applied to the chest, where the rubbing sound was heard.

I would just remark, with reference to this case, that you find here all the phenomena of rheumatic fever; and, in addition, the remarkable fact that a severe internal inflammation, involving the pericardium, and likewise, probably, the endocardium and the pleura, supervened *whilst the patient was in a state of salivation from the early administration of mercury.*

On the 12th we found our patient suffering rather less pain; she had also been refreshed with some sound sleep. From that day until the 24th she continued steadily improving: the pain in her joints diminished, and then ceased altogether; the pulse and respirations became less frequent, and the sweating diminished. The pericardial friction sound continued to retain its to and fro character, but shifted towards the apex of the heart, becoming less extensively heard and softening down. A pleuritic friction sound was heard for one day only, the 15th, over about the sixth rib on the left side.

On the 24th she experienced a return of pain in the left side of the chest, and also in the left shoulder. On the 26th we found her not nearly so well; her tongue was dry and furred; the pulse was feeble, and had risen to 140; she was restless and could not sleep. On examining her chest, we found extensive dulness on percussion in the pericardial region; the heart-sounds were distant and indistinct; the to and fro sound had ceased, and in its place we heard a distant systolic murmur; a pleuritic rubbing sound was detected also over the lower part of the right lung in front. These changes in the physical signs seemed to point, clearly enough, to a considerable effusion of fluid into the pericardium. The quantity of opium taken, which had been reduced on the 18th to half a grain three times a day, was again increased to a grain; a blister was also applied over her heart.

With the exception of the extension of the pleuritic rubbing over nearly the whole of the right lung in front, some oppression of breathing, and a change in the joints affected, there was no marked alteration in the condition of our patient or in the physical signs until the 29th: all oppression of breathing then ceased; there was no longer extensive dulness on percussion over the heart; the former to and fro rubbing sound returned, loud and harsh, and was heard pretty extensively over all the front of the chest, but most marked towards the base of the heart and under the clavicles; the pleuritic rubbing was still heard on the right side. From these changes the reabsorption of the effused fluid was at once inferred.

By the 3d of February the general co
had much improved: both

sounds had disappeared, and the systolic bellows sound, which had been masked by the to and fro rubbing, was again distinctly heard. On the 5th there was distinct evidence of a circumscribed patch of pleuritic inflammation on the left side. From this time, however, a rapid and steady improvement took place, and by the 10th the pulse had fallen to 70, and improved in tone and quality; all pain and abnormal sounds had ceased, excepting a mitral systolic murmur, which continued audible at the apex of the heart, and there was now some return of color in her lips and cheeks. This was after about five weeks' residence in the hospital.

The opium was now discontinued, and a tonic plan of treatment commenced, the patient taking a grain of quinine three times in the day, which was subsequently changed for three grains of the ammonio-citrate of iron.

A rapid convalescence, after so severe an illness, was hardly to be expected; our patient accordingly remained in the hospital for some weeks, and, although on the whole improving, had occasional accessions of pain and swelling in some of her joints.

I will now give the history of three other cases which I have selected as average examples of the course and duration of this disease under the plan of treatment which I now pursue; and with these I must bring this lecture to a conclusion.

CASE III.[1] (Vol. xlv, p. 103.) Matthew Baldwin, aged 29 years, a laborer, accustomed to liberal potations of beer. His father, he says, suffered much from rheumatic gout, but his own health has been generally good.

For about four weeks previous to his admission on September 21st, 1854, he had been generally ailing, with feverishness, headache, disordered bowels, and loss of appetite. In the course of the last of those weeks there had been an accession of rheumatic pains in the ankle, knee, and hip of the left side, then of the right, with increasing severity. Finally, the day before admission, he was seized with pain in the right shoulder and præcordial region, with a sense of tightness and difficulty of breathing.

The first night after his admission, he slept little and perspired profusely. When examined, the following day, he appeared pale and ill, and distressed by shortness of breath; his tongue was furred, his skin warm and moist; the right wrist was the only painful joint. The urine, which was highly acid, and of specific gravity 1035, contained much lithate of ammonia. The pulse numbered 100, and the respirations 40. A distinct rubbing sound was heard all over the region of the heart.

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[1] The record of this case was kept by my clinical clerk, Mr. Goodall.

As his bowels were confined, he was ordered some of the hospital white mixture, consisting of the sulphate and carbonate of magnesia, and this was followed by frequent doses of the usual alkaline mixture, containing fifteen grains of the bicarbonate and five of the nitrate of potass in each dose. A blister was also applied over the heart.

His breathing appeared almost immediately relieved after the application of the blister. On the third night he slept well; and on the following day, September 23d, he had almost lost the pain in his chest; the respirations were much easier, and had fallen to 27 in a minute, his pulse to 98.

On the 25th there was an increase in the number of joints affected, the right knee, ankle, and wrist, being very painful; small blisters were therefore applied to them, and the blister to the chest was repeated. He was then sweating freely; his tongue was cleaner, bowels open, and appetite improved; the urine was clear, and had fallen in specific gravity to 1028; the pericardial friction sound still continued. The following day (26th) he was ordered two drachms of brandy every two hours, or six ounces daily.

There was no important alteration for some days. On the 2d of October the decoctum cinchonæ was substituted for water in the alkaline mixture, and he was ordered five grains of the pil. saponis comp., with three of calomel, in pill every night. On the 3d, the blister to the chest was repeated. On the 4th, his back was observed to be covered with sudamina. On the 7th, being the seventeenth day from his admission, the following note was taken: "He is now improving daily. Pulse 84, respirations 26; sleeps well, and does not sweat; the pain is confined to some of the muscles; a slight friction sound alone remains audible at the apex of the heart; tongue clean, appetite good, bowels open; he still continues the brandy."

After this he remained some weeks in the hospital, gaining strength, and improving much in health; there was a return of slight pain in the joints, especially the shoulders, for which iodine paint was applied. A tonic mixture of quinine and acid was substituted for the alkaline one.

CASE IV.[1] (Vol. xxix, p. 188.) Johannah White, a servant girl, 16 years of age, came under my care in the hospital on the 24th of January, 1850; for some months before her admission she had not had her usual good health. On the night of the 22d she awoke with severe pain in her back; this was soon followed by pain in her knees and shoulders, gradually increasing in severity.

On the 25th, the day after her admission, she was suffering from great pain in the right shoulder, with pain and effusion into both knee-joints; her skin was hot and sweating, the perspiration acid in reaction and smell; she had no appetite, was thirsty and sleepless; the tongue was thickly coated with a blankety white fur; pulse 120, respirations 36; urine very acid, and loaded with lithates. She at once commenced taking the ordinary mixture of the bicarbonate and nitrate of potass, with five minims of the tincture of opium every four hours. A blister was also applied above the left knee.

The next day, January 26th, the joints were much the same, and, in addition, the right wrist had become extremely painful, and much swollen; she had slept badly; her pulse still numbered 120, and respirations 36; urine the same.

On the 27th, the third day of the treatment, her pulse had fallen to 84, and the respirations to 30; she had slept well, her appetite had returned, and she was free from pain. The following day she had slight pain in the right shoulder, but continued otherwise improving.

On the 31st of January, the tenth day of the attack, she was quite free from pain; her tongue was clean; pulse 72; respirations 20. There was no relapse, and she was soon after discharged well.

CASE V. (Vol. liv, p. 140.) Deborah Monssey, a servant girl, 14 years of age; accustomed to much out-of-door work and exposure. About six days before her admission she was seized with rheumatic pains in her ankles; the knees were next affected; and, the following day, all the joints of her limbs.

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[1] This case was recorded by my clinical clerk, Mr. Monckton.

On admission, January 31st, 1857, her face was flushed; skin hot, but not perspiring much; features constrained; tongue covered with a thin white fur; bowels confined; pulse 126, and respirations 36. The joints most affected were the shoulders and knees. The urine deposited a dense brick-colored sediment. She complained of uneasiness, or slight pain in the præcordial region; on listening, we detected a distinct pericardial rubbing sound. Soon after, a soft systolic bellows sound, heard most distinctly over the base of the heart, was also observed. She was ordered to take two-drachm doses of the liq. ammon. acet. with three minims of tincture of opium every four hours. Six leeches were applied over the region of the heart, an alkaline wash to the lower limbs, and a blister above the most painful joints.

On the third day of the treatment, February 2d, the rubbing sound had almost ceased, the systolic bruit remaining distinct; she had sweated more freely during the night, and the limbs, on the whole, were easier. Her medicine was now changed for the mixture of the bicarbonate and nitrate of potass, and fifteen minims of the liquor morphiæ muriatis were ordered to be taken at night. On the fifth day, February 4th, the pain was almost confined to her wrists and hands; the tongue was cleaning in the centre; her countenance was natural; pulse 124; respirations 36.

She continued in the hospital for about a fortnight after this, improving, but suffering more or less from pains in the different joints, shifting about, and varying in intensity; she was also much troubled with nausea and vomiting. Her pulse steadily declined to 65; her tongue became quite clean; the urine clear and bright; and the bellows sound softened down.

Before I enter upon the description of the treatment of the disease, let me come to some understanding with you as to its nature; for we cannot adopt a particular plan of treatment without having some theory of the nature of the disease. Now, what is the most reasonable view of the pathology of this disease? I have not time to enter into the discussion of this question with you as fully as I could wish; and I must, therefore, be content with simply recounting to you the articles of my own creed upon this subject.

Rheumatic fever, then, I would say, is a state of high febrile excitement, induced by the accumulation of a peculiar morbid product, or *materies morbi*, in the circulation; and the other symptoms which accompany it are merely caused by certain local derangements and disturbances produced at those points whence its elimination from the system is taking place. This materies morbi is the result of a vitiated state either of primary or secondary assimilation, or of both, and the parts where it accumulates are just those which, while they are very vascular, and therefore contain a large quantity of the diseased material, present the least .obstruction to its escape from the circulation. These are the delicate synovial membranes of the joints, and the almost identical structures, the serous membranes—the pericardium, endocardium, and pleura, the air-cells of the lung itself, and even the peritoneum—parts where the bloodvessels are naked, or covered by but a film of membrane. These membranes, being largely supplied with rheumatic blood, pour forth into their cavities an enormous amount of their ordinary secretion, contaminated with the diseased material. Thus the synovial membranes become distended with a morbid synovia, which, instead of being alkaline, as it is in health, has a reaction decidedly acid. Thus, likewise, the skin is covered with profuse sweats, which are due to the irritation established in the sweat-glands by the morbid product; and the abundant fluid thus got rid of has, like the synovia, a marked acid reaction. The functions of the kidneys are, doubtless, similarly affected, and you get an abundance of lithic acid in the urine. But this morbid matter may escape likewise through the serous membranes, as it does at the synovial, at the lungs, or at the heart; and hence, at any of these places it may excite inflammation, and at all of them is disposed to do so; and it is evident that the more its elimination is encouraged and favored at the skin, at the kidneys, at the joints, and at the mucous membrane of the alimentary canal, the less likely are the other important parts to suffer—the less chance have you of pleurisy, pneumonia, pericarditis, &c.

I have thus given you an outline of, and illustrated the principal features, both essential and accidental, of rheumatic fever, and we have come to an understanding as to the pathology of isease. I must defer to another lecture some account of the ent which appears to me the most appropriate.

# LECTURE II.

## *On Rheumatic Fever.*

HAVING, in my last lecture, described and illustrated the prominent points in the clinical history of rheumatic fever, I must now proceed to that upon which I wish to dwell particularly, and which, indeed, is the main object of these lectures, namely, the treatment of that disease. Upon this subject there still exists a good deal of difference among practitioners; and as I have myself, after much inquiry, come to some decided conclusions as to the line of practice which should be pursued in these cases, and as they are confirmed to me by daily experience, I am anxious to bring the whole subject before you, and explain fully to you the principles which regulate my practice in the treatment of this formidable malady.

It is important that we should determine what are the particular objects to be kept in view in the treatment of diseases of an acute kind. They are these:—

1st. To relieve pain.

2d. To strike at the root of the malady.

3d. To cure our patient with as little trial to his constitution as possible, so that afterwards he may not be in a worse condition than he was before. We often hear in society such expressions as these: "I was always very well till Dr. So-and-so treated me for rheumatic fever, and he purged and bled me to such a degree, and treated me so violently, that my constitution could not stand it, and I have never been the man I was before." Now, let us endeavor to conquer this frightful malady, and let it be our boast that, when we have done so, we leave our patient a constitution unimpaired, at least by our remedies. In some cases it is not possible to accomplish this; the lungs may become affected, or the pleura, or the pericardium, or the endocardium, and so much organic mischief may be done in a short time as to leave important organs permanently damaged; still, notwithstanding these lesions, the general nutritive powers need not be materially injured.

4th. A good plan of treatment should aim at securing for the patient a short convalescence. I do not speak of a speedy cure, because that is, to a certain extent, implied in a short convalescence. At the same time, I must caution you against the so-called rapid cures said to be effected by the heroic treatment of rheumatic fever. If these cures are rapid, they leave a tedious and painful convalescence; indeed, it may be more properly said in such cases, that the treatment converts an acute into a chronic disease, rather than that it cures the former. Such a cure, if cure it can be called, is not what you should aim at obtaining for your patients; nor is that a *bona fide* cure of rheumatic fever unless the febrile and constitutional symptoms are subdued, the secretions re-established in their normal quality and quantity, the tongue rendered clean, and the joints relieved of their swelling and pain: if such a cure as this can be effected in a short time, not entailing a tedious convalescence, your patients will have good reason to be satisfied. Now I must tell you that I do not believe that a *bona fide* cure and a short convalescence are, in the generality of cases, really obtainable by the heroic modes of treatment; and I would add my conviction that it is not desirable to shorten very much the period of cure in this disease, as it is not likely that a sufficient elimination of morbid matters can be effected in a very short time. These, so-called rapid cures, are also apt to leave the patient very subject to relapse, which you should endeavor to guard against as much as against a tedious convalescence.

The most instructive way, as it seems to me, in which we can discuss the treatment of this disease, will be to enumerate the various methods which have been proposed for this purpose, and to point out the reasons for rejecting some and for adopting others. As many as seven different plans may be specified, of which I shall place last that which I am in the habit of following here, and which I call the *treatment by elimination.*

The first plan is that by venesection. It was formerly the prevailing opinion, and it is still, unfortunately, thought by some that, when called to a case of rheumatic fever, one had only to open a vein, and if he could succeed in taking away a sufficient quantity of blood, which, in many instances, it was laid down should be little short of one or two pints, that, by this large and rapid abstraction of blood, the disease may be cut short, and a malady, which ordinarily lasts some weeks, may be converted into one of a few days' duration. Frequently, not content with one large bleeding, those, who hold these views, will bleed a second, a third, or a fourth time, at short intervals, and in large quantities.

The chief advocate of this practice at the present day is Bouillaud, of Paris. Now, if you look through the record of cases, as given in his book, you will see that his patients, although some of the more urgent symptoms are apparently very quickly overcome, yet linger on in the hospital for a considerable period, suffering much from chronic rheumatism, and exhibiting an extreme anæmia, from which they but slowly, if ever, recover. This plan of treatment has been advocated by some English physicians, and among others by the celebrated Sydenham, who, however, in the latter part of his career, abandoned, or greatly moderated it; and, I am happy to say, the number of its supporters at present is very small. It is a practice from the adoption of which I would most earnestly dissuade you, as having the support neither of reason nor of experience, and as being fraught with the most dangerous consequences to your patients.

I could tell you of several cases in which a fatal result has been clearly produced by the adoption of this method of treatment, which, most probably, would have recovered completely had they been left alone, or treated by a milder method. One case in particular made a deep impression upon me. The subject of the case was a young and strong man, of great promise in his profession; he was seized with rheumatic fever, and one of the knee-joints was severely affected. On a previous occasion a similar attack seemed to yield readily to a very large bleeding, and the patient recovered. His medical attendant, naturally enough, determined on the second attack to adopt the same treatment which had seemed so successful before, and accordingly bled him very largely, and applied leeches to the inflamed joint. The result was violent delirium, and death by exhaustion in the course of about eight-and-forty hours.

The following case, treated some years ago [illegible] will serve to show what venesection, and the loss of blood by other means, can do, as well as what it cannot do:—

Case VI. (Vol. i, p. 260.) Charles Davis, a porter, 28 years of age, was admitted into King's College Hospital on the 12th of October, 1840. He stated that, about a week previously, after exposure to cold, he was seized with lumbago; that the pain afterwards left his back, and attacked his arms and legs.

On the day of admission (the 12th), the left knee and ankle and the right wrist were painful, and somewhat swollen; his pulse numbered 90. Fourteen ounces of blood were immediately taken from the arm, and a purgative draught administered; the blood drawn was buffed and much cupped, and the clot large. That night he sweated freely, but the severe pain prevented sleep. The following day (13th) the pulse was rather less frequent, 80, and a white fur on the tongue, so usual in acute rheumatism, was noticed. He was ordered to take a quarter of a grain of the muriate of morphia twice in the day. On the third day he was suffering severe pain in the left hip, and along the back of the thigh; the sweating had continued; his pulse had risen to 100, was thumping, full, and compressible. The morphia was discontinued; he was ordered two grains of the sulphate of quina three times a day, and was cupped near the painful hip to twelve ounces. The pain diminished after the cupping.

When he had been eight days in the hospital he was nearly free from pain in the joints, but complained of a pain in the præcordial region, where an indistinct bellows sound, following on the systole, was detected. He was cupped over the heart to six ounces. On the 12th day he complained of a sense of weight in his chest, for which a blister was applied, apparently with benefit. But the next day there was a pericardial rubbing sound heard in addition to the bellows sound; the pulse had risen to 96, and there was a return of pain in several joints, and a red blush over the left ankle. He was again bled from the arm to eight ounces; the quina was discontinued, and a pill, containing one grain of calomel and a fourth of a grain of opium, was ordered to be taken three times a day. For five or six days afterwards he continued suffering chiefly from wandering pains; the rubbing sound was persistent; the pulse about 96, full, and slightly thrilling. He had taken two colchicum draughts with morphia, in addition to the medicines above prescribed; but these were all discontinued on the eighteenth day of admission and the quina resumed.

The last report of him was made when he had been ill about five weeks, and in the hospital about a month. He was then complaining of slight pain in the chest, and had a throbbing pulse numbering 80, but was otherwise improving.

This patient remained another fortnight in the hospital, and was then discharged; but had hardly been away a fortnight when he had a slight fit of shivering, soon followed by pain and stiffness of several joints, and considerable swelling of the feet and knees. He waited a fortnight and was then admitted, for the second time, to the hospital, December 23d, 1840, with all the symptoms of rheumatic fever. He was immediately bled from the arm to sixteen ounces, ordered to take a purgative draught at once, and an ounce and a half of guaiacum mixture three times a day. Besides suffering pain in almost all his joints, he complained, for many days, of a constant painful sense of weight and tightness in his chest, for which he was cupped to twelve ounces on the third day of admission. At first the heart sounds were normal, but, on the fourth day, a slight systolic bruit was heard, and there was occasional hiccough. He continued taking the guaiacum mixture for a week without any decided benefit; at the end of that time the left shoulder and wrist were still painful, and the pain in the chest continued. A grain dose of the acetous extract of colchicum every six hours was then substituted for the guaiacum. On the fourteenth day from his second admission, a blister was applied to the chest, as the pain there continued; and on the twenty-first day there was some increased articular affection; the left wrist especially was swollen and red. Six leeches were applied to the wrist, ten grains of Dover's powder were ordered to be taken at night, and the colchicum to be omitted.

On the following, or twenty-second day, there was great præcordial pain, preventing him from lying down, with hurried respiration, and a slight pericardial friction sound. He was cupped, over the heart, to seven ounces, and a pill, containing three grains of calomel and half a grain of opium, was ordered to be taken every three hours. After the cupping he was in less pain.

and able to breathe more comfortably for two days, but his pulse continued up to 100, and the rubbing remained.

On the twenty-fifth day, there was a fresh accession of pain in the chest, with dyspnœa; he was therefore bled again to sixteen ounces; the blood was much buffed and cupped; the pain was less after the bleeding. The next day the pulse was 100, and the respirations 48, with considerable dyspnœa on any exertion; a distinct pericardial rubbing sound was heard near the sternum, between the third and fourth ribs, and over the anterior surface of the heart; a bellows sound was also distinctly audible at the apex. He was again bled to twelve ounces; the blood was not buffed.

During the two or three following days our patient continued suffering much pain in his chest, as well as in some of the joints; the rubbing and bellows sounds continued; there was considerable sweating, and sudamina made their appearance on the neck and chest. As we might have expected, he was now very pale and feeble, with a quick pulse, sleeping badly, and suffering from palpitation on any exertion. After slight ptyalism, the calomel was discontinued, and some Dover's powder given alone; three ounces of wine were also added to his diet, but soon changed for a pint of porter.

He continued very slowly improving for nearly three weeks, but at the end of that time, seven weeks from his admission, he had more pain again in his chest, with catching breathing, and a quick, throbbing pulse; a return of pain soon followed in the joints. A blister was applied to the chest, and ten leeches to the left knee and ankle. The pain then shifted to other joints; the right pleura became affected—there was pain in that side increased by deep inspiration, and a rough rubbing could be distinctly felt and heard; pain continued over the heart, and there was hiccough. When the pleuritic symptoms appeared, leeches were applied to the right side, and also to one hand; the next day he was cupped to seven ounces, and a sixth of a grain of the muriate of morphia was ordered every six hours.

He remained more than five weeks longer in the hospital, improving very slowly: he continued to have pain with a sense of dragging in the right side, the creaking sound also remaining audible. The morphia was soon discontinued, and the quina resumed; occasional blisters were applied to the right side. He was at length discharged better, but not free from pain, on the 30th of March, having then been ill sixteen weeks, and an inmate of the hospital, the second time, for nearly fourteen.

The case, however, does not end here; he was discharged only to be readmitted two months afterwards, having then been suffering for a month from a fresh attack of rheumatism. He was admitted on the 26th of May; the left elbow was then painful, and both ankles painful and swollen, with effusion into the sheaths of the neighboring tendons. When he had been in the hospital a day or two, he had an attack of acute pain in the lower part of the chest, on the right side, with dulness on percussion. Ten leeches were applied, and he lost the pain, but continued to complain of a feeling of oppression, for which a blister was tried. He again got better, and left the hospital, but the date of his discharge is not recorded. During the two former periods of his residence in the hospital he had been bled altogether five times, and cupped five, so that one hundred and ten ounces of blood had been taken, besides what was lost by leeches! I shall content myself with remarking upon this case, simply, that it seems to me one well calculated to show that bleeding, mercury, and colchicum are not all-powerful for good in the treatment of rheumatic fever.

This subject is so important, that I shall offer no apology for bringing before you a second case treated on a similar plan.

Case VII. (Vol. i, p. 228.) Elizabeth Freethy, a servant, 21 years of age, was admitted to the hospital September 9th, 1840, with rheumatic fever. She had then been ill three weeks, and attributed the commencement of the attack to having caught cold in a damp kitchen. She was first seized with violent pain in her back; her limbs soon became affected, and two days afterwards she had nearly lost the use of them, but she did not take to her bed until about three days before her admission.

When admitted (9th) she was ordered to take a dose of the hospital white mixture, and ten grains of Dover's powder at night. On the following day (10th) the aperient mixture was repeated, and she was cupped in the loins, but the amount of blood taken was very small.

On September 11th she was bled from the arm to the amount of eight ounces, and the Dover's powder was continued. On the

12th, the bleeding was repeated, and twelve ounces more of blood were taken; she was then ordered two grains of the sulphate of quina three times a day. On the 19th, twelve ounces of blood were again taken from the arm, and on the following day the quina and Dover's powder were omitted, and a small dose of the muriate of morphia substituted.

On the 25th she complained of pain in the region of the heart, and a slight bellows sound was heard. There was some effusion into both knee-joints; the pulse numbered 122; the tongue continued white and furred. Twelve leeches were applied to the chest over the heart. The next day she was perspiring profusely.

On the 27th, when she had been ill rather more than five weeks, and under treatment in the hospital nearly three, the following note was made: "She chiefly complains of her knees, which are very painful, and full of fluid, the left one especially, the skin of which is slightly red on the inner side, and very painful, even when lightly touched; pulse 112; no sleep." Six leeches were applied to each knee. On the 28th there was no material change; a systolic bellows sound continued audible; her pulse numbered 120; she still perspired profusely, and had not slept. Ten-grain doses of bicarbonate of potass, three times a day, in soda-water, were then ordered. On the following day there was some general amendment, and less pain, and by the 1st of October all feverish symptoms had subsided; her appetite was returning, and she could sleep better. The knees and ankles, however, continued painful, and somewhat swollen.

After this she made little progress; and on October 6th, when the last report was made before her discharge, the knees, though not swollen, were still painful, and her pulse continued as high as 98. She had then been ill very nearly seven weeks, and had been rather less than a month in the hospital.

In this case, again, you have a striking example in which the disease was of long duration, despite of free and repeated bleeding.

I think it but right to state, in justice to those who have adopted this practice, that patients so treated sometimes do very well; and the next case that I shall quote will furnish an example of this. At the same time I must caution you against allowing such a case to lead you to adopt a similar plan of treatment. That it might justly so influence you, it would be necessary to show that such favorable cases are not only as common, but more common, under an antiphlogistic plan of treatment, than when less violent remedies are used; and not only so, but that, among unfavorable cases also, those are most disastrous in which the abstraction of blood has been abstained from. Judging from no inconsiderable number of cases, treated on both plans, I am convinced that the opposite conditions obtain. I would go so far as to say that, even were we certain that venesection would produce the desired effect on the leading symptoms of the disease, we should yet hesitate ere we make use of a remedy which, in the *general* effect it may have, is often uncertain, and most perilous. In one case you may relieve your patient, in another you may send him to a premature grave; or in the same individual, in a first attack, you may obtain complete relief by this method, and in a second attack you may place his life in jeopardy, or subject him to a tedious convalescence.

CASE VIII. (Vol. i, p. 210.) Francis Barrett, a carpenter, 26 years of age, was admitted an in-patient of King's College Hospital, with rheumatic fever, on September 11, 1840. The attack began the day before with pain, redness, and swelling of the left foot and ankle, accompanied by feverish symptoms, great thirst, and heat of skin. The pain soon extended to the muscles of the calf.

When admitted he was bled to twelve ounces and purged. He gradually became worse, and by the 12th all his joints were affected and the pain severe. On the 14th he was still suffering severe pain in every joint, and also complained of some uneasiness about the heart; his pulse was observed to be intermittent, and occasionally there was a sort of double systole of the ventricle. He was then cupped, and sixteen ounces of blood taken; a mixture was prescribed, consisting of sulphate of quina with one-eighth of a grain of muriate of morphia in each dose. At night he perspired freely, and on the morning of the 15th he was in less pain. During that day, however, and the two following, there was no great improvement: the redness and swelling of the wrists remained; his tongue was coated with a white fur; there was profuse perspiration, and a high pulse of about 100.

On the 18th he was again bled from the arm to sixteen ounces, and the clot formed was much cupped and buffed. On the

the joints of the right upper extremity were still acutely painful, but his pulse had fallen to 88. On the 20th there was decided improvement, and on the 21st he was much easier: the swelling had disappeared from the right wrist, he had slept better, was sweating less, and his tongue was cleaning; pulse 68. The quina was repeated without the morphia.

He continued improving; and on the 24th, being the fifteenth day of the disease, he was free from pain, but weak; his pulse was 60, his appetite improving, and tongue clean. A chop and a pint of porter were now added to his diet, and we have no history of a relapse.

2. The second plan of treatment is that by moderate bleeding and diaphoretics. This may be called an "expectant" treatment; but it is more than that as regards the venesection; while in other respects it sufficiently merits the name. The advocate of such a plan will say—"When I am called in to a case of rheumatic fever, I think it advisable to commence the treatment by abstracting about ten or twelve ounces of blood, and then to give sudorifics and purgatives."

Now, the objection which I entertain to such treatment is this: that the routine abstraction of blood can scarcely be called *necessary* in any case, and that in many it is injurious. The tendency of rheumatic fever is to impoverish the blood, especially as regards that highly important portion of it, its coloring matter. All that bleeding really effects is to relieve pain (which, however, may quickly return) for a few hours, while it undoubtedly aids the bleaching power of the rheumatic matter, and, as I have observed in several cases, it increases much the tendency to a chronic rheumatic state, and consequently prolongs the convalescence. That bleeding in rheumatic fever is unnecessary, and that its omission diminishes rather than increases the tendency to certain internal inflammations, I am so convinced, that for several years I have not abstracted blood, in any way, in a single case of the disease. The treatment of rheumatic fever by the abstraction of blood, even in moderate quantity, but more especially in large quantity, appears to me to increase the danger of internal effusions into the pericardium and the pleura, and also into the synovial sacs of the joints (*vide* Case VII). Under this treatment we also meet with the most violent and troublesome cases of delirium, which, under other methods, either does not occur, or is developed in a form sufficiently easily controlled. I am very much disposed to believe that this treatment predisposes to pericarditis and endocarditis; and that, if these affections occur in a case in which venesection has been freely practised, they are much less tractable than when you have to deal with them in a patient who has not suffered from loss of blood.

3. A third plan is that by mercury. Some recommend that calomel and opium should be freely administered until salivation is produced. The great objection to this treatment is, that it is an attempt to cure one fever by setting up another, and, in some respects, a worse: even supposing the original disease succumbs, your patient comes out of his rheumatic fever with loose teeth, ulcerated gums, and all the painful and offensive concomitants of ptyalism. Now, I say, that, under such circumstances, the remedy is nearly as bad as the disease; and, moreover, it does not in the least guard the patient against what may be termed the accidents of his malady—those severe internal inflammations—pericarditis, endocarditis, pneumonia, pleuritis, peritonitis. I have more than once seen pericardial inflammation supervene while the patient was in a state of salivation, of which the Case (II) of Sarah Green, detailed in the first lecture, is a good example. When we consider how differently various persons are affected by a mercurial course, and how much some suffer from it, even if given in small quantity, it would seem highly inexpedient to adopt this plan of treatment, for it assuredly offers no prospect of effecting either a speedy cure or a speedy convalescence, much less both together.

It is worthy of remark that rheumatic patients sometimes exhibit a distinct tolerance of mercury, and are with difficulty salivated. The following case is an instance in point, affording, at the same time, but little encouragement to the supporters of the mercurial treatment, and yielding no evidence of the anti-rheumatic power of mercury.

CASE IX.[1] (Vol. xv, p. 70.) John Smith, a lad, 15 years of age, after a day of unusual exertion, November 29th, 1845, went home and to bed very much fatigued, and awoke the following morning to find his knees and ankles painful, hot, and swollen. He rested all that day, and then returned to his work in much pain, with feverish symptoms, and general indisposition. On December 3d, the fourth day of the disease, he could not leave his bed, and the next day was sent to the hospital. When admitted the same joints were still affected; pulse 100; respirations 38. The knees and ankles were wrapped in cotton wool, and he was ordered ten grains of Dover's powder in saline mixture three times a day, and a dose of the hospital white mixture.

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[1] Reported by my clinical clerk, Mr. Sturt.

On the night of the 5th (sixth day), he perspired freely and rested well; but on the morning of the 6th (seventh day) he felt pain all over his chest, with difficulty of breathing, and an unusual throbbing at his heart; his pulse had risen to 110. On the 8th there was no improvement; the pain in his chest continued; his face wore a constrained appearance; he was breathing 36 times a minute, and his pulse was 104; some roughness of the first sound was heard towards the base of the heart; the urine gave a copious precipitate of lithates. He commenced taking a grain of calomel with each Dover's powder, the aperient mixture was repeated, and a blister applied over the heart.

On the 9th, the tenth day of the disease, there was a general improvement, though the same symptoms remained. This continued ten days, the articular affection subsiding, while our patient's general condition improved, and his pulse fell to the natural standard, so that on the 18th he was ordered two grains of the sulphate of quina three times a day. On the following day, the 19th, he was not so well, complaining of pain in his back and shoulders, palpitation, and difficulty in deeply respiring. A blister was applied to the chest, the quina discontinued, and the Dover's powder and calomel resumed, the dose of the calomel being increased from one to two grains. On the 20th he was better again; but a mitral systolic bellows sound was heard, and the blister was repeated, and afterwards dressed with a mixture of equal parts of mercurial and savin ointment. The improvement continued, and on the 26th he left off the calomel, after having taken it, with but one day's omission, for eighteen days without salivation. He then resumed the quina; and by the 7th of January was well enough to leave the hospital, after a residence there of about five weeks.

He had not been away a week when he was again attacked with pain in his chest and palpitations, together with pain and swelling of the left knee; he was therefore readmitted on the 17th of January, and a blister applied to his chest. On the 18th, he commenced taking the alkaline mixture of the bicarbonate and nitrate of potass, which I now so constantly prescribe, with five grains of Dover's powder at night. On the 19th the blister was repeated, and three grains of calomel added to each Dover's powder; although better, he was still suffering from some articular pains, from palpitation, and from symptoms of general disorder, with a rapid pulse of about 110. A rough systolic bellows sound was audible both at the base and apex of the heart on the 21st; and on the 24th a slight pericardial rubbing sound was also heard at the base. By the 27th he was free from pain and feverish symptoms, but his breathing remained quick, and the rubbing sound was still heard.

The last note was taken between a fortnight and three weeks later, on the 14th of February. He had then been taking citrate of iron for some days, but was evidently much exhausted, as his pulse, which was 88 when reclining, rose to 108 on standing up. We have no record of the date of his discharge.

4. Another plan of treatment which has been proposed is by colchicum and by guaiacum. These drugs, but especially colchicum, have long been considered to possess a specific influence over rheumatic and gouty affections; and it has been laid down that the rheumatic condition will be subdued in just such proportion as you get your patient under the influence of the colchicum, somewhat in the same way as quinine exercises a specific influence on ague. Now I think it requires only two or three cases to prove to a candid mind the fallacy of this doctrine. I myself have frequently given this remedy the fairest trials, but I could never discover any effect from it sufficient to entitle it to the character of a specific. That it is capable of exerting a remarkable influence, as well for evil as for good, on gout, I do not deny; but even this must be admitted with considerable limitation; it is certainly far from exercising any similar or analogous influence in rheumatism, whether acute or chronic. The effect of guaiacum has also been supposed to be [illegible]
to that of colchicum; but it has [illegible]

Both these medicines, when given in large doses, purge, and, in such doses, I have no doubt they may do some good, on the principle of eliminating the morbid material by the alimentary canal; but unless you give them in such quantity as to produce colliquative purging, you do but little towards cutting short the disease; and if you do give them in these large doses, you produce a degree of prostration and debility which is sometimes more dangerous than the disease, and you leave your patient to linger through a tedious convalescence. Colchicum given in small doses produces no good effect in rheumatic fever, according to my experience; on the contrary, I fear that in some cases it has a prejudicial influence on the nervous system, making it more irritable and susceptible of impressions, and rendering the patient more obnoxious to the various accidents that are liable to occur in the course of the disease.

5. Treatment by opium. This plan of treatment has been lately revived by a very able physician, Dr. Corrigan, of Dublin. It has much to recommend it, and, on the whole, you will find it extremely serviceable in practice; but I do not recommend it alone: its great value consists in relieving suffering, and soothing the nervous system, while it promotes diaphoresis. The opium is given in large and frequently repeated doses, care being taken not to produce too much narcotism; but upon this point, in general, there is not much need for fear, as there seems to be in the generality of patients a remarkable tolerance of opium. Our patient, Elizabeth Stocking, whose case I have described in the last lecture, was ordered on the 23d a grain of opium, to be given every three hours, in addition to half a grain of the muriate of morphia, which she had previously been taking at night: in forty-eight hours she thus took sixteen grains of opium, exclusive of the morphia, yet her pupils were not at all contracted, nor was she in any degree narcotized. The effect upon her has been most beneficial: her nervous excitement has been calmed down, and her pain materially relieved. The same plan was pursued in the case of S. Green, also detailed in the last lecture: she had one grain of opium every three hours from January 8th to February 10th, excepting for one week, in which the dose was reduced to half a grain; the same tolerance of the remedy was observed. It will not, however, do to employ this plan alone;

it should be conjoined with other treatment. I do not recommend it by itself.

I may here adduce another case in illustration of the benefit to be derived from opium, where there is much disturbance of the nervous system with restlessness and delirium.

CASE X.[1] (Vol. xxxiii, p. 55.) George Rinning, a tailor, 45 years of age, of intemperate habits, and with some history of rheumatism in his family, obtained admission to the hospital on February 12th, 1851, with severe pain and swelling of the knees, ankles, and wrists, and profuse sweats. He stated that these symptoms came on, with loss of appetite and confined bowels, ten days previously, after exposure to wet and cold. Blisters and cotton wool to the joints, nitrate of potass with liquor ammoniæ citratis every four hours, and a dose of hospital white mixture, were prescribed.

On the 13th, the day after admission, a slight systolic bruit was heard at the base of the heart; he was suffering rather less pain, but his tongue was coated, dry, and brown; the perspiration and urine were very acid; his thirst great, and appetite bad. Throughout the night he was delirious, and could get no sleep, and continued forgetful and talkative the next morning. The bellows sound was more marked on the 14th, and a blister was applied to the chest; the nitrate of potass was discontinued, and a grain of opium given every four hours. At night there was much less wandering, and towards the morning of the 15th he slept. During the day he was drowsy, and his pupils contracted, but there was much less pain, and general improvement. The opium was discontinued during the day, but repeated at night.

The case afterwards followed an ordinary and favorable course. By the 5th of March he was able to sit up, and was discharged on the 8th.

6. A sixth plan of treatment, proposed long ago by Dr. Haygarth, consists in giving bark in large doses, for which, more recently, the less bulky sulphate of quina has been substituted. Now just imagine the state in which the pathology of a disease must be, when measures so completely at the opposite extremes of our

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[1] This case was reported by Mr. E. Liddon.

therapeutical resources are advocated for it—as venesection, to the amount of two or three pints, on the one hand, and large doses of quinine on the other; some would even give as much as five or ten grains two or three times a day. Now I have tried both methods of treatment, and I approve of neither; but if I were tied down to one or other of them, I should not hesitate to choose that by bark. In cases where the sweating is colliquative, and the urine copious and pale, with abundant precipitates of *pale* lithates, I have seen great good done rapidly by the use of quinine; but I am not prepared to advise you to adopt this treatment from the beginning, because it tends to check secretion, and so may favor the development of internal inflammations.

7. The seventh and last mode of treatment that I shall mention to you is, that which you have seen me adopt frequently at this hospital, namely, *the treatment by elimination.* I give it this name, in order that you may keep well in view its main object—to promote the elimination of morbid matter by the various emunctories, and also that you may bear in mind the view of the pathology of the disease upon which it is founded.

It is probable that *the materies morbi* in rheumatic fever is lactic acid, or some analogous agent. We know that the natural emunctory of this is the skin. Many chemists maintain that it will also escape by the kidneys; and if it ever does so, perhaps this is more likely during rheumatic fever than at any other time. Again, since vitiated digestion is apt to produce it in undue quantity, and it therefore is formed abundantly in the stomach, there is every reason to think a certain proportion of it may be carried off through the alimentary canal. The indications are, then, to promote the action of the skin, the kidneys, and the bowels; to use antacid remedies; and to give large quantities of fluid for the free dilution of the materies morbi, and to supply the waste caused by the drainage from diaphoresis and diuresis.

The best way to promote the action of the skin is by opium, especially if you combine with it nitre and ipecacuanha. For this purpose I sometimes use a compound which resembles the original Dover's powder, in containing nitrate of potass, instead of sulphate of potass, as prescribed in the compound ipecacuan powder of the Pharmacopœia. Our usual prescription is one grain of opium, one grain of ipecacuanha, and five grains of nitre; this must be given every two, three, or four hours, according to the urgency of the symptoms, and the need the patient has for opium. This drug quiets the nervous system, and procures sleep, and with the ipecacuan promotes sweating; while the nitre acts upon the kidneys, and the ipecacuan may exercise some influence on the liver.

The best alkali on the whole is the bicarbonate of potass, which may be given in large and often-repeated doses—a scruple or half a drachm every third hour. Sometimes the acetate of potass answers very well in similar doses, and many physicians much prefer it to any other alkaline salt.

Next you must give purgatives to such an extent as to keep the bowels in a loose state, taking care not to carry this treatment so far as to weaken your patient, or worry him by obliging him to be frequently moved in and out of bed. You will find it advantageous to use an alkaline purgative; and there cannot be a better medicine for this purpose than our hospital nostrum—the white mixture containing magnesia and sulphate of magnesia. Sometimes you may give the potassio-tartrate of antimony with advantage; but as it is a depressing remedy it is seldom advisable to use it.

But while we are thus alkalizing our patient, and giving internally sudorifics and diaphoretics, ought we not to attend to the state of the joints? The diligent physician will tell you by all means to attack them at once: but there is such a thing as "nimium diligentiæ" in physic as well as in other matters. Many will say, the best thing you can do is to leech a painful and swollen joint: I formerly tried this practice extensively, but for some time past I have not done so, as I generally found it either useless or injurious. You may apply leeches, and in a short time after you will find the pain and swelling removed, and you may be disposed to say, "Here is a proof of their efficacy;" but wait twenty-four hours, and then you will generally find the pain and swelling as bad as ever, and the joint in just the same condition as before. Now apply leeches, and you will probably fail to give any relief. You have by the first application relieved the pain for a time, but you have produced no permanent good; you have rendered the disease more erratic, and less amenable to subsequent treatment. Frequently when you leech a joint, the pain and swelling subside, but its fellow becomes swollen; leech it, and the swelling and pain return to the original joint. Nothing is more important to avoid, nor more troublesome if not prevented, than the erratic tendency of the rheumatic state. It will fly from joint to joint, and in pursuing it with leeches you only drive it out of one joint into another. I am satisfied that leeching the joints favors this erratic tendency.

I am not prepared, however, to advise you to neglect the local treatment of the joints. When they are much swollen and painful, you may give great ease to your patient by enveloping them in a large quantity of the soft carded cotton—commonly called *cotton-wool.* Over this you must wrap a sheet of oiled silk, so as to cover in the wool completely, taking care to have no part ot it exposed. By this air-tight covering you keep the joints in a complete vapor-bath; and when you come to remove the oiled silk and wool, after twelve or twenty-four hours, you find the wool completely saturated with moisture, which generally is strongly acid. You have seen this in Elizabeth Stocking's case. We find the plan so generally useful, that it is adopted in the hospital in nearly every case: it affords great relief, supports and keeps the limb steady, and at the same time promotes sweating. I may just mention, that this plan of enveloping the joint in wool and oiled silk is also very beneficial in gout.

In a few, and only a very few, cases, I have found the pain aggravated by the heat which this mode of wrapping generates; and in cases where it is desirable to keep down the sweating, it is not advantageous to carry this plan beyond a day or two.

The best additional local treatment is that by blisters of small size, applied on or near the affected joints; they are very useful both in acute rheumatic and acute gouty joints. I shall refer to this subject again. (*Vide infra.*)

You perceive that all the means employed in this mode of treatment tend to elimination, and to the relief of pain: the opiate sudorific affecting the skin; the nitre and alkaline salts acting on the kidneys; the purgatives on the mucous membrane of the bowels; the wool and blisters on the joints.

During this treatment, while you allow your patients the liberal use of simple diluents, you must give a fair amount of nourishment from the first; and I think this may be best supplied by a small quantity of good beef tea, given frequently throughout the day.

Often you will find it useful, and always when there is a tendency to delirium, to give stimulants, such as brandy or wine. A good example of the benefit from the timely use of stimulants is afforded by the following case:—

Case XI.[1] (Vol. xxix, p. 236.) John Wilks, æt. 24, was admitted February 9th, 1850. He had been attacked, about a fortnight before, with pain in his left great toe, soon followed by swelling; at the same time he began to suffer from languor, loss of appetite and thirst; he sweated much at night, and noticed that his urine was high-colored, and deposited a deep red sediment. Pain and swelling soon followed in most of the larger joints.

When admitted, he was sweating profusely, and the perspiration had the peculiar odor and acid reaction so constant in acute rheumatism; the right ankle, the knees and wrists were painful and swollen, the least motion of the last causing excruciating pain; his pulse was 100; his tongue coated with a white fur. Fifteen grains of the bicarbonate with ten of the nitrate of potass, and five minims of tincture of opium, were given every four hours; and two blisters were applied to the wrists. A decided diminution of pain in the wrists followed the application of the blisters; at the same time the pulse became more frequent, and continued above 110; in other respects the symptoms remained the same on the 10th and 11th. On the night of the 11th he became delirious, and the delirium recurred the following night; he was therefore ordered half an ounce of brandy every two hours. There was no return of delirium. By the 15th, there was general improvement; on the 13th and 14th he had suffered from diarrhœa, but this had ceased; he was free from pain; his tongue moist and cleaning; he slept well, and his pulse had fallen to 84 and improved in tone.

He continued to make favorable progress until the 5th of March, on which day he had a slight relapse, but soon recovered, and was discharged cured on the 16th.

I have many more remarks to make on other points in the treatment of rheumatic fever; but must content myself now with having given you an outline of the eliminatory mode of treatment, and reserve my further observations for another lecture.

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[1] Reported by Mr. Dickinson.

## LECTURE III.

### *On Rheumatic Fever.*

In my last lecture, gentlemen, after having passed in review six different methods of treating rheumatic fever, I particularly recommended to your attention one which we have been in the habit of using here, the object of which is to promote as much as possible the elimination of morbid matters from the system through the natural emunctories—through the skin, through the kidneys, through the bowels. I advised you to use opium freely, potass and nitre, to give alkaline purgatives, and to relieve the pain and swelling of the joints by enveloping them in cotton wool, surrounded by oiled silk. And all this I ventured to recommend to you in preference either to the plan of treatment by venesection, or that by colchicum or guaiacum, or that by calomel.

Now, it may sometimes happen that you will have to deal with a patient who is unable to take opium. What are you to do under these circumstances? There is no reason why you should change the general plan of treatment—you may still give sudorifics—and if your patient will bear sedatives, you can give hyoscyamus, or hop, or extract of lettuce. But it will, I believe, very seldom happen that, in this severe and painful malady, patients will be unable to bear opium in some shape or other; and the benefits to be derived from the proper use of this drug are so great that you ought to try it in various ways, and in different preparations, before you abandon it altogether. I think that practitioners often fail in obtaining all the good effects of opium from being too timid in the use of it, giving it in too small a dose, and employing it in a vacillating manner; you must give it in a large dose, not less than a grain, frequently repeated, taking the state of the pupils as your guide to encourage in or deter from proceeding with it. You will of course proceed with

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great caution if you find a very contracted pupil in addition to some degree of narcotism. Before you abandon the use of opium, remember that you have a great variety of forms in which to prescribe it; you have, among others, the compound camphor tincture, which is often borne when the other preparations fail; the acetate and muriate of morphia; Mr. Battley's liquor opii sedativus; and a preparation introduced by Mr. Squire, the solution of the bimeconate of morphia, which may be given in the same doses as laudanum; and codeine, as prepared by Pelletier in Paris.

Again, it may happen, and this is by no means of unfrequent occurrence, that the swollen and painful state of the joints does not yield to the cotton wool and oiled silk only, or that the heat, which that application generates, cannot be tolerated by the patient. What further treatment of the joints will you pursue? I have no hesitation in advising you to apply blisters; and I would recommend you to use every means in your power to get them to rise well. I do not think it advisable to apply *large* blisters; on the contrary, they are injurious, and their use is to be deprecated. The plan I generally follow is this: I order a small mustard cataplasm to be applied to the affected joint, and to be kept on for half an hour to redden the skin; after its removal the skin is to be carefully washed and dried, and the blister may then be applied; you must not let the size of this exceed that of a crown piece. It is better to apply two or three small blisters in rapid succession, and to different parts of the joint, than one large blister. After the blister has risen well, if the swelling of the joint subsides quickly, as it very frequently does, you may let the blister heal as fast as it will; but if the swelling has not subsided, then you had better cut away the cuticle completely, and promote a free discharge from the blistered surface by dressing it with stimulating ointments. Some prefer to apply the blister above rather than over the joint.

You need not be afraid to apply blisters in the early stages of the rheumatic inflammation of the joints. I believe the dread which some physicians had, and have, of applying blisters near inflamed parts—as near an inflamed lung, or pleura, or pericardium—is due to their having used blisters of too great a size. I have applied them very early to rheumatic joints in numerous cases, and always with more or less advantage, provided the blisters have not been too large. A very large blister is very apt to do mischief, and augment the inflammation of the joint; but a small one, varying in size from that of a crown to a half-crown, is almost invariably beneficial. When a very copious effusion has taken place into a joint, the plan of applying two or three small blisters in succession, at different parts of the joint, provided the first should fail in getting rid of the effusion, is productive of the best effects.

I have seen excellent results from the application of blisters to gouty joints, even in the most acute stage. A discharge of a large quantity of serum from the vessels of a gouty joint has all the good effects of the abstraction of blood from it, without any of the evil consequences of that mode of treatment.

You must exercise a proper caution not to carry the sweating or the purging process too far with your patients. It is impossible to lay down precise general rules on this subject: the state of the patient's pulse, his countenance, the mode in which he expresses his feelings, will sufficiently indicate the condition of his general powers to enable you to judge whether you are going too far or not. On this point of the treatment I would advise you to take as your motto—*ne quid nimis;* neither too much sweating, nor too much purging, nor too much opium. I shall not caution you against too much bleeding, but I deliberately, and without hesitation, advise you to omit that from your practice altogether in the treatment of this malady; and I do this from a large experience and observation of its little efficacy for good, and its great liability to do serious mischief.

All the world now knows how important it is in acute rheumatic cases—and, I would add, even in chronic also—to pay close attention to the heart. You should watch it from day to day, and from the very commencement of the attack; and if you find the smallest indication of a departure from its normal mode of action, attack it specially and at once. I say you should watch the heart from the very first moment the patient comes under your charge; for the cardiac symptoms are apt to come on very early, and in some instances they precede the articular affection. In our patient, Elizabeth Stocking, the cardiac symptoms must have developed themselves very early, as they were already well marked on her admission into the hospital.

The circumstances that will denote to you that the heart is beginning to suffer, are,—irregularity of the pulse *in any way*, either as affecting its force or its rhythm—*i. e.*, whether the intermission be partial or complete: or its becoming suddenly quicker or slower. Should any of these signs present themselves, you should at once institute the most minute scrutiny into the physical signs of the heart's action, and if you should find the slightest indication of a rubbing or bellows sound, you may infer that either the pericardium or endocardium, or both, are beginning to suffer. Disease of the endocardium is especially to be feared if the bellows sound is mitral systolic, *i. e.*, if it be heard most distinctly over the apex of the heart, and beneath the left scapula behind, and accompany the first or systolic sound: under such circumstances, you may be sure that the endocardium is suffering, and that some portion of the mitral valve is implicated in the lesion. If, however, the systolic bellows sound be heard most distinctly over the *base* of the heart, and along the course of the great vessels, and is therefore aortic, you must not at once infer that this is a sure sign of the existence of endocarditis, affecting the aortic valves: you must bear in mind that an aortic bellows sound may, and very frequently does, arise from an anæmic state of the system. I have already told you that the rheumatic state tends to diminish very much the proportion of the coloring matter of the blood, even in patients who have not been bled, or otherwise roughly treated. The rheumatic state itself, then, by bleaching the blood, may give rise to aortic and even venous murmurs. How much more likely to be produced is the condition favorable to these murmurs when bleeding has been practised. You must be very careful not to fall into the mistake of treating an aortic murmur as due to endocarditis, which is really the result of the already bleached state of the blood. Such a mistake is not unlikely to be made, as the diagnosis is difficult between the anæmic murmur and that from aortic obstruction; and you can readily understand how an antiphlogistic process, especially if it included bleeding, would make matters infinitely worse in a case where the murmur was simply of the anæmic kind. The more you proceeded with such a treatment, the more, of course, would the conditions favorable to such a bellows murmur be developed, and the louder it would become. The following points will [illegible] you in deciding upon the endocardi

First, if the sound come on very early in the disease; second, if it be rough in character; third, if it be not accompanied with venous murmur; fourth, if the patient has not yet displayed much anæmia; lastly, the probability of an endocardial affection is much increased if the murmur have been ushered in with some disturbance of the heart's action, such as I have already referred to.

It is important, especially with reference to prognosis, to keep in view that the mitral valves may be affected, first, so as to induce valvular imperfection; and, secondly, so as not in any degree to impair the function of the valve.

If the deposit of lymph take place on the auricular surface of either or both curtains of the valve, then you will have valvular imperfection: the curtains will not meet exactly, and a fissure will remain, of larger or smaller size, through which more or less of regurgitation will take place into the auricle at each ventricular systole.

But if the deposit take place on the *ventricular* surface of the valve,—and it generally does so on the ventricular surface of its inner curtain,—then you have no disturbance of valvular function. In both cases, however, you have a systolic bellows sound; and in both cases that sound is best heard at the apex of the heart.

How are you to distinguish the one from the other? If the bellows sound be purely regurgitant, its position is strictly at the apex; it becomes in a marked way faint as you proceed to the base of the heart, and it is distinctly audible beneath the left scapula; and, in addition, the sign pointed out by Skoda exists, namely, a marked intensification of the second sound. If the bellows sound be not regurgitant, you hear it well up to the base of the heart; you hear it only feebly, or not at all, at the left scapula, and there is no intensification of the second sound. I may add, that, in this latter case, the heart's disturbance, and the sufferings of the patient, are, in a marked manner, less than in the former.

It must also not be lost sight of, that a bellows sound, now present, may be the result of a previous attack of rheumatic fever. You must rely mainly upon your knowledge of the patient, or upon such a history of his previous state as you can pick up by inquiry among his friends and relations, to distinguish whether the endocarditis is new or old. Of course, certain symptoms of newly-come-on heart disturbance would favor the former view; but I know of no physical sign or sound which aids the diagnosis.

In the treatment of the heart affection, I am in the habit of acting upon much the same principle as in that of the joints; and I trust to free vesication and the promotion of a copious discharge, serous or sero-purulent, as the local treatment. I shall describe to you the plan I am in the habit of following, and which we have used with the most satisfactory results in Elizabeth Stocking's case. On the first indication or suspicion of heart affection, a large sinapism, made with flour of mustard and hot water, is applied over and beyond the region of the heart; this is to be kept on as long as possible. After its removal, and after the skin has been properly cleansed, put on a blister of good size; and you must be guided as to the dimensions of it by your opinion of the extent to which the heart is affected. You need not be afraid of large blisters here, as in the treatment of the joints, because the inflamed organ is much more distant from the surface than the synovial or other articular tissues.

If you pursue the plan which I have thus pointed out, and have drawn a large quantity of blood to the surface by the long-continued stimulation of mustard, you will generally succeed in producing very free and large vesication, from which you may obtain a considerable quantity of serum,—or rather, I should say, of *liquor sanguinis*, for the fluid of the blister is serum containing more or less fibrine. If you examine the fluid from blisters, especially when the skin has been previously irritated by mustard, you will almost invariably find that it contains more or less of fibrine. In very many instances, if not in all, the coagulated fibrine disposes itself in a membranous layer in immediate juxtaposition with the deep surface of the elevated cuticle. On removing the cuticle slowly and cautiously, the serum will not flow away; it is still retained by a very complete, but soft, moist, and almost spongy membrane. This is coagulated fibrine, which has entangled in it a large number of the white corpuscles. How these latter escape from the bloodvessels, or whether they are not the result of the organi[illegible]
the liquor sanguinis, I cannot pretend
however, that blisters will take a

its dissolved elements, and perhaps the rudiments of the white corpuscles. By blistering you take away that part of the blood which is the great agent in the development of new formations, and these are what you have to guard against in the cardiac inflammations. Moreover, by blistering you spare that most important part of the blood, the coloring matter, which seems especially valuable for preserving the nervous functions in a state of integrity, and which is no less important for maintaining the healthy action of the heart.

But some of you will say, "What! do you advise us to lay aside that which has so long been regarded as the sheet-anchor in the treatment of inflammations,—namely, bleeding; and not only general bleeding, but topical bleeding likewise? If we are neither to cup nor to leech in pericarditis or endocarditis, what security, then, shall we have against the progress of inflammation,—against the formation of excrescences on the valves,—against ulcerative or suppurative processes being established in the heart, destroying its valves, and infecting the blood?" I am quite aware that the doctrine which I recommend for your adoption is likely to be regarded as extremely heterodox by many; but I believe the number of those who think so is daily diminishing. In the treatment of the cardiac affections which accompany rheumatic fever, you have two objects to keep in view; the first is, to check the morbid process completely, or to restrain it from producing such changes as may prove destructive to the tissues, and consequently to the mechanism of the heart: and the second, to obviate liquid effusions which may distend the pericardium, compress the heart, and so embarrass its actions, as well as the respiratory movements, as to prove seriously detrimental to life. Now, with regard to the first point, there can be little doubt that bleeding will not stop or prevent the formation of those fibrinous concretions which are so apt to form upon the valves. The formation of these concretions is in a great measure mechanical, and in certain states of the blood they would form around or upon any opposing material, just as fibrine will coagulate round the bunch of twigs by which blood is beaten as it flows from a vein. In this rheumatic state, the contractile tendency of fibrine is apparently increased, as is shown by the uniform formation of a tough buffy coat in the blood removed from rheumatic subjects; there is also a considerable increase in the number of white corpuscles; the buffy coat is formed of these two constituents, and the constancy of its formation denotes a tendency in these two elements to separate from the other elements of the blood in the rheumatic state. Doubtless, a disturbed state of the nutrition of the serous membrane or the endocardium, or of certain parts of them, precedes the formation of fibrinous deposits upon them; and this disturbance of nutrition is caused by the accumulation of the rheumatic matter in the vessels of the part. The effect of this is analogous to, if not identical with that produced by a blister on the vessels of the skin, which I have just now described to you. The liquor sanguinis transudes through the parietes of the bloodvessels, and the plastic matter coagulates upon the surface of the endocardial and the pericardial membrane, forming there a substance identical, or nearly so, with the buffy coat of the blood. In the endocardium, which is in contact with the blood as it flows through the heart, this layer of plastic matter forms a nucleus, around which fibrine from the blood which flows over it may coagulate.

Now, if this be a correct account of the manner in which the plastic concretions develop themselves in pericarditis and endocarditis—and I believe it is that which is most consistent with our present improved knowledge of the blood and of inflammation—it is evident that the object of the practitioner should be to prevent the development of that altered state of nutrition which *precedes* the fibrinous formation, or to arrest it prior to the pouring out of the fibrine. Will bleeding do this? I think our experience of the effects of bleeding upon the joints ought to convince us that it will not: for bleeding certainly will not remove the rheumatic state from them; for, however it may relieve for a short time, by diminishing hyperæmia, or by some influence on the nervous system, the flow of blood speedily returns with as great, or greater activity than before. I apprehend that the state of the joints and that of the heart is as nearly as possible the same, the difference being that the nature of the synovial secretion offers a much greater physical impediment to the formation of fibrinous or plastic concretions in the joints than exists in the endocardium or in serous membranes.

And I would put another question: Will bleeding cut short that state of blood which is so favorable to the formation of the plastic deposits? To this I answer likewise in the negative. Among the best of the modern researches upon the relative quantities of the elements of the blood in various conditions of that fluid, are those of Becquerel and Rodier. What do these observers say as to the influence of bleeding upon the blood? Why, that it considerably diminishes the red particles, that it very much augments the proportion of water, and that it affects but little or not at all the fibrine; thus, in short, you get a thinner liquor sanguinis, holding in solution the same, or nearly the same amount of fibrine. In other words, you get a state of liquor sanguinis very favorable to transudation, and therefore very favorable to plastic formations.[1]

If, then, bleeding will not stop the inflammatory state which creates the undue determination of the blood to the pericardial and endocardial surfaces, and if it will not prevent the plastic formations, but rather favor them, surely it is not the remedy for pericarditis and endocarditis. And if the effects of venesection be,—as beyond all doubt they are,—to diminish all the solids of the blood but the fibrine, and to augment the water, surely the employment of this treatment is fraught with the greatest danger of creating liquid effusions into the serous and synovial sacs, which are so exposed to the action of the rheumatic matter.

These are, as concisely as I can put them before you, the theoretical grounds upon which I object to the practice of bleeding, whether local or general, for the cardiac affections of rheumatic fever. And my experience confirms me in the belief that the practice of bleeding is altogether unsatisfactory in its remedial results, and prejudicial in its consequences. I have likewise learned by experience that the practice of abstaining from this mode of treatment is perfectly safe, and tends to the best results. By the general plan of elimination—locally, by blisters,—generally, through the sweating and other augmented processes of secretion—you divert the rheumatic matter very freely from those great central and highly vascular organs which we are so anxious to protect from mischief.

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[1] The analyses of Dr. Christison show an increase of fibrine under bleeding; and those of Dr. Beale show the same fact to a remarkable extent, in the blood of a dog bled on four successive days to the extent of six ounces each day. *Vide* Todd and Bowman's Physiology, p. 312, vol. ii.

Besides the local treatment that I have prescribed, you must, when the heart or any of the great internal organs is affected, still keep up the influence of opium upon your patient, whereby you secure a powerful means of keeping down excessive action of the heart, of calming the nervous system, and of promoting cutaneous elimination.[1]

You will bear in mind that both pneumonia and pleurisy are very common complications of rheumatic fever; but for the treatment of these affections I have nothing to add here to what I have said respecting the treatment of the cardiac affections. The treatment of both should be exactly the same, *mutatis locis.*

There is a very formidable complication of rheumatic fever, respecting which I must say a few words. I allude to the delirium which is apt to manifest itself in the course of the attack; sometimes with thoracic inflammation, sometimes without it. It is very important that you should be prepared for this symptom, and that you should understand its nature, and its proper mode of treatment; it is not in itself a dangerous symptom, unless the practitioner fails in taking the precautions which are rendered imperative by its occurrence.

The delirium of rheumatic fever sometimes comes on gradually, the patient having been a little talkative and wandering for two or three nights; sometimes it comes on quite suddenly. In its general characters it resembles delirium tremens—generally, however, exhibiting less of the nervous tremor which belongs to intemperance. The patient is restless, busy, talkative, picking or pulling the bed-clothes, frequently rising in bed, and wanting to get out of bed, reaching out his hand as if to catch hold of some object before or behind him, and sometimes—a most unfortunate symptom—obstinately refusing to take either food or medicine.

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[1] In the impression of this Lecture, which first appeared in the London Medical Gazette, for October 20th, 1848, the following paragraph appears:—

"I know that there are many physicians who speak lightly of the remedial powers of mercury in these rheumatic affections. But I confess to you that I am not prepared to give up the dogma of Dr. Farre, that mercury is opposed to, and breaks down, plastic formations. Still I must admit, and this is satisfactory for patients who may be prevented by idiosyncrasy from the use of mercury, that I have seen patients do extremely well without having taken a single grain of that medicine."

I have omitted this paragraph from the text in this edition of the Lecture, because my subsequent experience has led me more and more to agree with those who repudiate the necessity for the employment of mercury in these affections; and I feel myself justified in declaring my belief, that under the treatment described in the text, the results are more favorable, and altogether more satisfactory, as regards the future of the patient [illegible] d. At the same time, I am quite ready to [illegible] them is suitable to [illegible]

In many instances, as I have already said, this delirium ushers in pericarditis, pleurisy, or pneumonia; frequently, however, it occurs after one or other of these maladies has set in, and sometimes it occurs without them. It has, therefore, I think, no necessary connection with these internal inflammations, although it frequently accompanies them.

Now, what is the nature of this delirium? It used formerly to be viewed as a metastasis of rheumatism to the brain, and to be treated antiphlogistically. I have treated some cases in this way, and on this hypothesis, and I have had the opportunity, in consequence, I believe, of this treatment, of examining the state of the contents of the cranium in a few such cases. I can therefore assure you that there is no more inflammation, either of the brain or its membranes, in these cases, than in delirium tremens. The membranes are perfectly free from abnormal deposit, the pia mater is pale, and the gray matter of the convolutions remarkably so, and the subarachnoid fluid is increased in quantity. These signs indicate not only that the brain has been imperfectly supplied with blood during life, but that the vascular pressure upon it is less than it ought to be, and that, consequently, an increase of the subarachnoid fluid has taken place.

When, then, we consider the circumstances in which the brain is placed in these cases, we cannot wonder at its functions being disturbed. In the first place, the organ is supplied by a depraved blood—a blood deficient in its most important staminal principle, its coloring matter—a blood infected with an abnormal material, the rheumatic virus, whatever that may be; and a watery blood, which is the more apt to exist, if the patient, as is very often the case, have been treated by sanguineous depletions. Such a blood is ill suited for the proper stimulation of the heart, and consequently it is not propelled by that organ with its proper force, although the rapidity of the heart's action may be much increased; and if the heart be inflamed, there can be no doubt that the effect of that inflammation will be to weaken still more the propelling power. Hence, in cases of this kind, the brain is feebly furnished with a blood, poisoned, poor in coloring matter, and abounding in water.

I have met with a few cases in which the patient, having evinced previously little or no delirium, has become rapidly comatose, with dilated pupils, and sunk quickly. And it sometimes happens that patients who have been actively delirious will suddenly fall into coma and die; and sometimes they die suddenly, while making some effort beyond their strength, in the midst of their delirious ravings. The state of the kidneys may have some influence in determining the mode of death in those patients who pass quickly into coma, as we know that defective action of those organs so often exercises a baneful influence on the brain.

A case occurred to me in private practice, which shows how rapidly rheumatic fever will sometimes run through all its stages, and exhibit all its phenomena,—articular, cardiac, cerebral,—notwithstanding the active and early treatment by calomel, colchicum, purging, &c. I will give you the case briefly.

Case XII. A student of one of the universities, aged 22, had complained for two or three days of pain in the left foot. This became suddenly very much aggravated at the railway station, as he was starting for his college, on the 17th of April, 1855. He went to a hotel, and put it into mustard and water, but proceeded next day to his destination. The rheumatic affection then extended to all the large joints; there was free sweating, and numerous sudamina made their appearance. He was treated by mercury, colchicum, liquor antimonialis, liquor ammoniæ acetatis, and latterly Dover's powder. I was telegraphed for to see him, and found him, on the evening of April 27th, extremely ill: much purged,—passing watery stools, apparently from the liquor antimonialis; he had a full throbbing pulse, and soon became very restless and delirious. I saw him again at midnight, and detected a friction sound over the heart. The delirium had increased, with a comatose tendency; but he could still be roused, and then recognized me and others about him. I ordered him half a drachm of the bicarbonate of potass and a grain of opium every three hours, and a small quantity of brandy. In the night the delirium increased, and he refused to take food or medicine; his breathing became catching; pulse

120; and soon after nine o'clock the next morning he died, eleven days from the supervention of the acute symptoms.

If I were to treat such a case from the beginning, I should employ opium at once; possibly, also, liquor ammoniæ acetatis; or, more probably, large and frequent doses of bicarbonate of potass, wool or blisters to the joints, beef tea, and brandy or wine freely. Purgatives should be given carefully, but not so as to cause colliquative purging. Such a treatment would husband the strength, and enable the patient to resist the influence of a large accumulation of the rheumatic poison, such as must have been present in this case.

You will find a valuable collection of cases of delirium and other disturbances of the nervous system, in connection with rheumatic cardiac affections, in Dr. George Burrows's interesting and most valuable work on Disorders of the Cerebral Circulation. The evidence which Dr. Burrows has adduced in that work should teach us, that whenever we meet with a case of delirium, especially of rheumatic delirium, we should diligently explore the region of the heart, and watch the condition of that organ most carefully from day to day.

But this delirium, as I have before said, has no *necessary* connection with the heart affection—at least, with endocarditis and pericarditis—for it occurs in cases of general gout, in which there are no such heart affections as those in rheumatic fever, and the delirium of gout resembles precisely that which I have described to you as belonging to rheumatic fever.

I have seen, indeed, this delirium in persons of strongly marked rheumatic or gouty diathesis, accompanied by all the signs of rheumatic fever—the sweats, the furred tongue, and the lithic urine, and not only without cardiac, but even without articular affection.

I may make this further remark, before I refer to the mode of treating this delirium, that what I have seen of it has strongly impressed me with the belief that it is much more apt to occur after bleeding, and in weakly subjects, than when depletion by bloodletting has not been employed, or in sthenic cases. It is also often an indication that your patient is being reduced too much by sweating, or purging, or some other means.

The development of this delirium should be, as I have already remarked, a warning to the practitioner to look out for cardiac or other internal inflammations, as pneumonia or pleurisy, or even peritonitis—which sometimes, although rarely, occurs in rheumatic fever—if such have not been previously detected. But it should likewise be regarded as a signal of distress, denoting that the powers of the constitution are unequal to the severe trial through which the patient is passing; and he should immediately come to the patient's aid, and make arrangements for having him constantly watched by competent nurses or other attendants, taking care that the patient shall never be left alone. If he have been sweating freely, that must be checked; the amount of bed-clothes may be reduced; if his joints have been enveloped with wool, it must be removed. In like manner, any other too free evacuation must be stopped, as purging, or the too copious discharge from a blister. Nourishment must be given very frequently, but in small quantities, so as not to embarrass the stomach; and this should consist of beef tea, arrow-root, milk; and it will be always necessary to conjoin with this wine or brandy, or porter, when that has been an habitual beverage, also to be given in small and carefully-adjusted quantities. If the patient be wakeful, sleep must be procured by the free administration of opium. These are the points to which you will have to direct your most watchful care. Provide against your patient being allowed to exert himself beyond his strength; remember that it is in this state that patients often die suddenly by syncope, and be careful to nourish and support them well. Eschew all local treatment to the head; even the application of ice is calculated to do mischief, by depressing the heart's action.

When, however, the patient evinces a marked tendency to coma, then of course you will not use opium. I would advise you to shave the head, and to counter-irritate it and the back of the neck, by sinapisms first, and afterwards, if you find it necessary, by blisters, pursuing at the same time those measures for the support of the patient which I have already pointed out, and which, you may be assured, are not less necessary in the comatose cases than in those in which active delirium prevails.

The two following cases afford interesting examples of the more severe forms of nervous symptoms—delirium, convulsions, and coma—as they occur in the course of rheumatic fever; and the post-mortem examinations are instructive, as pointing out the non-inflammatory nature of, the brain affection. I bring them before you, with the view of illustrating the real nature of this delirium.[1] The treatment was not such as I should adopt now. It was on the whole antiphlogistic (so called), although moderately so; negative, as I believe, so far as regards any good effects; but injurious, if not directly, yet by excluding other means which would have done good.

CASE XIII. (Vol. xii, p. 162.) The first of these cases is that of Maria Edwards, a servant, seventeen years of age, whose health had been previously good. On the 17th of October, 1844, she was seized with rigors, followed by a reaction, with pain in her limbs; at the same time she lost her appetite, became very thirsty, and suffered from headache and giddiness. On the following day both ankles became hot, swollen, tender, and painful; subsequently other joints became painful, and she was admitted on the 25th, the ninth day of the attack, with all the symptoms of rheumatic fever. Both knees and ankles were then tender and painful, but not much swollen; the pulse was 120; the skin hot and perspiring; tongue furred. A systolic bellows sound was heard all over the præcordial region, and there was a slight catching pain in that situation. The urine was dark-colored, and loaded with lithates and phosphates. The joints were wrapped in cotton wool and oiled silk, a blister was applied to the chest, and some Dover's powder ordered to be taken every fourth hour. On the day after admission blisters were applied to both knees.

By the 27th there was little alteration; she had no sleep at night. On the 28th she became delirious. For several days the same symptoms continued with but little variation, delirium recurring at night.

On the 31st (the fifteenth day) a distinct to-and-fro rubbing sound was heard over a great extent of the cardiac region. On the 1st of November, despite of two grains of calomel with Dover's powder and a grain of digitalis every fourth hour, with forty minims of Battley's solution of opium at night, the delirium, which had hitherto been confined to the night, was prolonged into the day. On the night of the 1st she was very restless, wandered much, and would get out of bed. Three ounces of wine were administered, and a little sleep procured, but the delirium soon returned, and about noon on the 2d forty minims of the liquor opii sedativus were given; her pulse was then 124, weak and compressible; the rubbing sound remained; her pupils were contracted; she was drowsy, and evidently much affected by the opium; she also suffered severely from headache, for which ice was applied, apparently with benefit.

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[1] For a full discussion of the pathology of this and other forms of delirium, see the Lumleian Lectures, delivered at the College of Physicians, 1850, and published in the London Medical Gazette of that year.

On November 3d the delirium had ceased, and her pulse had fallen to 96, and improved in tone; the three ounces of wine were continued during the day, and thirty minims of the liquor opii sedativus were given at night. For several days our patient continued better, nearly free from pain, and sleeping more quietly at night; the rubbing sound being still audible. On the 8th her gums were found tender and white; the saliva was slightly increased, but no fœtor was observed; the calomel was reduced.

On the 11th (twenty-sixth day) there was an unfavorable change, marked by a return of pain in the spine, shoulders, and right side, by restlessness and rambling talk at night, and by a weak and rapid pulse, numbering 140. These symptoms seemed to call loudly for supporting treatment; the daily supply of wine was therefore increased from three to five ounces (an insufficient quantity), and ten minims were added to the opiate at night. Some rest and comfort followed, and thus encouraged, we pushed the treatment a little further, giving half a drachm of the aromatic spirits of ammonia three times a day in camphor mixture, a grain of the sulphate of quina as often, and one ounce more wine. The blister to the chest was repeated, and afterwards dressed with mercurial and savine ointment. Though otherwise a little better, she continued to pass noisy, restless nights. The rubbing sound softened down, and nearly disappeared, making a loud systolic bellows sound more plainly heard.

On the 16th there was a fresh accession of præcordial pain, and a loud to-and-fro rubbing returned. The quina was doubled, and given every four hours with ten minims of tincture of opium.
The record of the 18th is as follows: "She only slept two h
last night, and not at all for the twenty-four hou
Pulse feeble, fluttering, 140; skin cool."
delirious, chattering and singing, and go

her pulse was 140, small and weak; her pupils contracted. Five ounces of brandy and some arrow-root were now given in the course of the day, and, to humor her fancy, a little fish was allowed; a grain of the muriate of morphia was given at night, and the quina was increased to four grains. In the evening the foot of the bed was raised, that her head might be lower, and a quieter night followed. The next day brandy was regularly administered every half hour in arrow-root. The last note was made on the 21st, after her death: "She was wandering all night; her pulse weak, quick, and fluttering. At seven o'clock this morning she changed for the worse; the rhonchus of the dying was heard; and, despite of brandy, administered at intervals through the night, she died at half-past ten this morning. Nitric and sulphuric æther were also given, in camphor mixture, through the night."

The examination of the body was made twenty-seven hours after death. The brain was found healthy, but the gray substance pale; there was no effusion on the surface or within the ventricles.

The lungs were congested, but otherwise healthy.

The opposed surfaces of the pericardium were adherent throughout by a layer of lymph, a quarter of an inch thick, but soft behind, and containing some fluid. The pleura, where in contact with the pericardium, was adherent, but there were no adhesions elsewhere. Warty excrescences of lymph were found on the margins of the aortic and mitral valves.

Here was a case which exhibited very clearly the natural course of the acute rheumatic disease with its complications. It was not likely that any part of the treatment would have materially modified the phenomena. And while we found unequivocal marks of intense pericardial inflammation with abundant plastic deposits, there was not a sign of anything to indicate inflammation either of the substance or of the membranes of the brain. That organ was pale, poorly supplied with blood, and resembled the brain of an animal bled to death. Again, let me remind you that there was none of that serous effusion on the surface of the brain to which many attribute so prominent a part in the production of the phenomena of delirium and coma. These effusions, indeed, we now know are *results* of the diminished size of the brain which follows its imperfect supply of blood, and its impaired nutrition, and as they do not exert any undue pressure on the brain or any part of it, they produce no symptoms during life.

CASE XIV. (Vol. xii, p. 57.) The second case is that of a single woman, Martha Mitchell, 34 years of age, who was admitted into King's College Hospital on the 18th of June, 1844, with her third attack of rheumatic fever. She stated that her general health for many years had been far from good; that she had suffered, she believed, from attacks of acute inflammation of the liver, with pain in the right hypochondrium and shoulders, and dyspeptic symptoms; that since her last attack of rheumatism, seven years previously, she had suffered from palpitation and dyspnœa on slight exertion.

One evening, more than a week before her admission, she went to bed unwell, with pain and stiffness in the right hip, and a feeling of chilliness, and awoke in the morning with pain in all her joints, especially in the knees and ankles, which subsequently became red and swollen; her appetite had completely failed, she had great thirst, and towards evening shivered violently. Two days afterwards, she was suddenly seized with palpitation and dyspnœa. The articular affection continued up to the time of her admission; she also perspired much towards night; the bowels became confined; the urine scanty and very dark.

When admitted, the joints of the arms, as well as the knees and ankles, were affected. There was pain in the præcordial region, and a systolic bellows sound was heard over the base of the heart and in the course of the aorta. Dover's powder, with nitrate of potass, was given every four hours, and the joints were wrapped in cotton-wool.

The next day, the 19th, the joints were less painful, her tongue covered with a yellowish-brown fur and red at the tip and edges, her pulse 120, and respirations 34. As there was still præcordial pain, a blister was applied, and two grains of calomel added to each powder.

The case goes on as follows: "11 P.M., June 19th. Having continued up to this hour in the same state, complaining of little or no pain, the physician's assistant was called to her, and found her delirious, talking incoherently, and the delirium panied with hallucinations; pulse somewhat increased in frequency, 128, weak and compressible; her skin hot and perspiring.

"She was ordered thirty minims of the liquor opii sedativus immediately. She slept after taking the opium; but at two o'clock A.M. the physician's assistant was again sent for, in consequence of her having had a convulsive fit affecting all her extremities. He found her lying on her back, her pupils very much contracted and insensible to strong light; pulse 132, weak but regular; her head hot, but the forehead perspiring; the respirations were 30, and of a croupy character. She was quite comatose. She had a return of the convulsions, screamed out, and died."

The body was examined thirteen hours after death. The following is the account from the case-book: "The body was very exsanguineous externally: the lips blue; the skin of the face and arms much freckled. The head, chest, and abdomen were examined.

"The vessels of the pia mater were not more than ordinarily injected except on the left side—(hypostatic?). There was no fluid in the arachnoid or subarachnoid cavities; the surface of the arachnoid membrane was, however, moist. On dividing the hemispheres horizontally, the vascular pink points were rather numerous, but no trace of disease was discovered.

"There was no fluid in the pleura, but adhesions around the apex of each lung, where there were also one or two caseous tubercles; the lungs were elsewhere healthy.

"The pericardium was almost universally adherent, and the adhesions were organized. Besides these, however, there were traces throughout of fresh inflammation; there were many flakes of soft lymph, and collections of a few drops of fluid here and there, with the surface much injected. The edges of the mitral valve were much thickened; all the other valves were healthy.

"The liver was large, and broke down readily under pressure; its convex upper surface was covered with a distinct, though thin layer of apparently condensed cellular membrane, with one small patch of lymph. The remaining abdominal viscera were healthy."

In the first of these cases, there was no evidence of any abnormal condition of the brain or its membranes, excepting, indeed, a slightly anæmic one. In the second, the congestion of one side of the pia mater was probably mechanical, and due to the gravitation of the fluid to the most depending part after death. The increase in the number of vascular pink points observed in the substance of the brain was probably connected with the mode of death,—in convulsions, which we well know congest the brain.

The next case is worthy your attention, illustrating as it does all the phenomena of rheumatic fever, the accompanying delirium, and the benefit likely to result in such cases from the early and liberal use of opium and stimulants, as I have recommended to you.

Case XV.[1] (vol. xxxiv, p. 166.)—George Gough, a footman, 19 years of age, of temperate habits, and usually enjoying excellent health, was taken ill, five days before his admission on the 10th of February, 1852, with general lassitude and aching of the limbs. The pains soon became localized in the joints with swelling; he also had pain in the left side of the chest.

On admission, the ankles, knees, and wrists were swollen and painful, and the hip and shoulders also; perspiration was profuse and acid; the urine acid, high-colored, and full of urates; his pulse was 104, and respirations 30; his tongue white and furred; he had lost his appetite, and could not sleep. On examining the chest, there was a slight systolic bellows sound heard at the apex of the heart. He was ordered some of the usual mixture, containing fifteen grains of the bicarbonate with five of the nitrate of potass, and five minims of laudanum, every four hours. The joints were wrapped in cotton-wool and oiled silk, and a mustard plaster was applied over the heart.

During the next two days there was not much alteration, and he passed sleepless nights, although the tincture of opium in the medicine had been increased to ten minims. The urine continued loaded with lithates and phosphates, and its specific gravity was about 1030. A blister was applied to the left ankle, and mustard plasters to each hip. The medicine was now given every four hours, and the laudanum increased to twenty minims.

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[1] For the record of this case I am indebted to my clinical clerk, Mr. Pearl.

As his bowels were confined, he was ordered five grains of calomel, followed by a dose of hospital white mixture.

On the 14th he was in less pain, and sudamina were observed scattered over his chest. That evening a to-and-fro rubbing sound was first observed over the base of the heart; this continued, and the next day a blister was applied, and strong mercurial ointment used in the dressing; his pulse remained as high as 126, and the respirations 40. On the night of the 15th he was for the first time delirious. On the 17th the whole body was covered with sudamina, and as the restlessness continued, he was ordered a night draught, containing half a drachm of tincture of opium; three grains of carbonate of ammonia were added to each dose of the mixture, and he began taking one ounce of wine every three hours (a quantity which, I think, was not adequate to the demands made upon the nervous power). That night, however, he was very delirious, trying to get out of bed and leave the ward, without any corresponding increase in the severity of the other symptoms, there being, on the contrary, a general improvement, excepting that the pulse remained 120. On the following day he was ordered a pill, containing two grains of calomel and a quarter of a grain of opium, with each dose of the mixture. On the 19th the delirium was not confined to the night, but he continued muttering to himself in the day, unless aroused or spoken to. The opium in his medicine was increased by five minims, and he took it every three hours; mercury also was rubbed into the axillæ.

On the 20th the delirium still continued, and he seemed unconscious of what was passing around him; his pupils were contracted; pulse 116, respirations 36. The mixture was omitted and five grains of carbonate of ammonia were given every two hours, together with half an ounce of brandy in beef tea every hour; the pill was continued every six hours. The favorable effect of this active stimulation was almost directly apparent: that same night he was much quieter, and slept a little, and on the morning of the 21st his pulse had fallen to 104, and the respirations to 26; some swelling of the knee-joints, and a pericardial rubbing-sound, still remained. He again had a comfortable night, and the next day, the 22d, his pulse was 96 and respirations 34. On the 23d he was going on most favorably: as his urine was alkaline, twenty-minim doses of chloric æther were substituted for the ammonia, and the brandy was reduced. On the 24th he was free from pain and progressing favorably; it was then just a fortnight from his admission, and about eighteen or nineteen days from the commencement of his illness.

After this he remained in the hospital about a month, regaining his strength under a course of tonics and good feeding; a rubbing-sound, and also a faint bellows-sound, continued to be heard for some time over the heart; there was also some chronic swelling of the knee.

I have already told you that you must be careful to carry out this general plan of elimination with the closest attention and regard to the powers of your patient's constitution. I allude to this subject again, for the purpose of mentioning to you a sign which has, over and over again, proved most valuable to me, in leading me to pursue an altered course of treatment. When the patient has begun to pass pale urine, in good quantity, either without precipitate, or with a greater or less quantity of pale lithates, you will almost invariably find that he will be the better for a more generous treatment, even although the articular affection still continue troublesome. You may give him ammonia, or quinine and sulphuric acid, and in many instances you may give wine or brandy; and I have been astonished at the rapidity of the progress of cases under this altered treatment; patients, whose symptoms had been stationary for two or three days, have, under the circumstances and treatment I have described, become convalescent in little more than forty-eight hours.

The plan of treatment which I have now recommended to you, does not contain any new remedy, nor does it profess to point to any summary method of treating rheumatic fever; it is merely the application of old and well-appreciated remedies to the treatment of this formidable malady, in furtherance of a certain determinate object,—that of eliminating morbid matter, at various points and through different channels, from the current of the circulation. Since I have adopted this mode of treatment I have much more rarely met with these
the disease,—pneumonia, pericar
so formidable to both the pat

severe form which I used to do under a more depleting treatment; and when such severe cases do occur in the hospital, they are generally persons who have suffered from a depleting treatment prior to their admission, or who have been thrown into a very reduced state from other causes. Again, I find that under this treatment the duration of the disease does not exceed from ten days to three or four weeks, and that relapses, which were very frequent under the treatment by bleeding, are of rare occurrence under this. Now it was formerly the dictum of an eminent physician, "that the only cure for rheumatic fever is *six weeks*." By this he meant that the disease would take its course, that time was its only cure, and that this time was not less than six weeks. But I should not attach much importance to a plan of treatment which failed to get patients into a good state in a much shorter time than that. Our patient, Elizabeth Stocking, whose case has been a severe one, and who has had pericarditis and slight delirium, has been in the hospital now just eleven days, and had been ill three days prior to admission, and you see that she is convalescent already. She has lost every rheumatic symptom; all the pains in her joints have ceased; her tongue is clean, and I have no doubt that in two or three days more she will be struck off the sick list altogether. And, as the last, though not least, advantage of this treatment, there is no fear of those unpleasant consequences which are so prone to follow in the wake of this disease: there is no fear of a tardy anæmic convalescence, for her blood has been spared; nor of a state of chronic rheumatism, for there is every indication that the whole of the morbid material has been eliminated from her system.[1]

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[1] The last note made before this patient left the hospital is dated June 17th, and is as follows: "She is improving in health and strength: appetite good: sleeps well."

I must add here an allusion to the plan of administering acetate of potass as suggested by the late Dr. Golding Bird, and large and very frequent doses of bicarbonate of potass as put in practice by Dr. Garrod. Both these physicians aim at making and keeping the urine alkaline. There is nothing in this treatment which militates against that which I have advocated in the foregoing lectures, and I may add that the free administration of alkalies is usually a very valuable practice, according to my experience.

# LECTURE IV.

## *On Continued Fever.*

Gentlemen: I wish to-day to call your attention to a case of common continued fever, with enteric disease, also called typhoid fever, which we have had lately in Rose Ward, and which we have watched with great interest, and not a little anxiety, for some days past. The case ended fatally, and for this reason I am the more desirous not to let it pass without some observations upon it. And I shall take this opportunity of giving you the following piece of advice: never shrink from analyzing and carefully thinking over the cases which prove fatal under your care, with a view to inquire, whether by a little more care you might not have been more precise in your diagnosis, and whether you might not have been more watchful in your treatment, or have adopted a more promising course. Such an inquiry, if faithfully pursued, involves an amount of self-examination which, in course of time, cannot but redound most beneficially to the character of the practitioner.

It is a doctrine supported by our best physicians and highest authorities, that you cannot cure a fever; that is, that you cannot cut it short: you can guide it through its several stages, you can support the patient's strength, uphold his vital powers, until the influence of the poison is worn out, and combat any accidental affections which may arise in the course of the treatment, such as diarrhœa, pneumonia, &c.; and by such careful management you may save the patient, by preventing him from dying by exhaustion, and you may shorten his convalescence considerably.

This is a doctrine to the truth of which I have for many years given my full assent, not only as regards typhus and typhoid fever, but also with respect to other fevers,—those, for instance, connected with the exanthemata. And although many, from time to time, have professed by some heroic method, as [illegible] very early, to cut the fever short, and thus to convert, what would otherwise have been a tedious and painful illness of three or four weeks, into a short attack of a few days, yet I have failed to convince myself, either by experience or reading, that any such important discovery has as yet been vouchsafed to us, as one calculated to destroy the venom of the typhus poison, and to check its ravages.

All the cases in which it has been said that typhus has been cut short, as by a very large bleeding at the outset, or by free vomiting, or by some other means, are fairly open to the strong suspicion, if not the charge, of erroneous diagnosis. It is plain, if you think on the subject but for a moment, that without an exact diagnosis, this question of the early curability of typhus cannot be settled. Now, those who have seen most of this and other maladies know best how difficult, nay, how impossible it often is, in the first week or ten days, to predicate with certainty of this or that case, that it is typhus fever. And, therefore, if you deal candidly with yourselves and others, you must not affirm that you can cut short and cure typhus, unless you have the most unequivocal evidence that the cases in question have been examples of that disease.

If these views be correct, you will perceive the necessity, when you come to treat a case of this nature, of not wasting time in trying this expedient and that medicine, but you will apply yourselves to provide for the due care and watching of your patient, and the careful administering to his wants and necessities. In this respect the poor, who are inmates of our public hospitals, have often a great advantage over the patients we have to treat in private practice; for here we have trained attendants, always ready, experienced in the management of cases of this kind, and accustomed to obey orders. In private practice, we are too often obliged to trust to the timid and inexperienced nursing of relatives and friends, or perhaps of servants already over-burdened with other duties; or, if we do succeed in overcoming prejudices, and in inducing the friends to procure the assistance of a nurse, it is too often the case that she is accustomed only to act as a lying-in nurse, and has no experience in fever cases. I would gladly read for you here the remarks of the late Dr. Graves, one of the greatest authorities on the subject of the pathology and treatment of fevers, on the choice of a nurse in cases of this kind; but I must content myself with referring you to the first volume of his valuable work on clinical medicine, where you will find them in the ninth lecture.

CASE XVI.[1] (Vol. xxxii, p. 75.) And now for the particulars of the case. The patient was a man, named John Gavin, 32 years of age, a large, bony man, of strong build. He lay in Rose Ward. He is a printer, and had just come from Edinburgh to look for work in London. His illness probably commenced in Edinburgh, and developed itself immediately on his arrival in London. It is often extremely difficult to fix precisely the day on which a fever began, partly from the imperfect recollection of patients, and partly because the symptoms often develop themselves so insidiously and gradually, that the patient cannot note exactly the time when he really began to be ill; he feels for many days languid and out of sorts, but is still able to get about, and, unless some such prominent symptom as rigor has occurred, it is impossible to name one day more than another on which the fever began. Now, what we gather is this,—that on or about the 9th, as he was leaving Edinburgh, he caught cold, of which he has no other evidence than the existence of great languor and weakness, with a strong sense of fatigue upon the slightest exertion. On his arrival in London, he found himself quite unequal to the task of looking out for work, and unable to follow his business if he had succeeded in securing employment.

All this looks very much as if he had caught the infection in Edinburgh, where, we know, fever is always more or less rife among the lower orders; its period of incubation being the day or two before he left that city, and the first few days after his arrival in London. During the first week of his arrival in town the sense of languor increased, and he felt very ill. On the 16th of January, 1851, sore throat came on, and he was attacked with several severe rigors, succeeded, on the 17th, by increased debility, vomiting, headache, and tinnitus aurium. On the 18th these symptoms had increased in severity, and his friends stated that he became stupid, and appeared as if drunk, and at times he wandered a little. It was, then, on the 16th, that the more decided symptoms of fever had developed themselves, although we cannot doubt that the poison had already begun to work in his system at least seven or eight days before that date.

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[1] Reported by my clinical clerk, Mr. J. H. Sylvester.

He was admitted into the hospital on the evening of the 19th of January. On the 20th, the following report of his condition that day was entered in the case-book: "The patient is very thin and weak; has a dull, vacant look; is delirious, incoherent, and it is not without great difficulty that answers to questions can be elicited from him; he is, however, very quiet, and lies chiefly on his back; respiration hurried; crepitation audible all over the posterior surface of both lungs, especially at their bases; the tongue is dry, but not coated; slight sordes on the lips and teeth; the abdomen slightly prominent and tympanitic; has had one loose motion in the night; no spots are observable; pulse 130, very compressible; respirations 44. He was ordered half an ounce of brandy with beef tea every two hours, and five grains of the sesquicarbonate of ammonia, with half a drachm of chloric ether, in an ounce and a half of water, every six hours, and turpentine stupes to be applied freely to the back."

On the 21st, his symptoms had not changed, and the pulse was 128; the respirations 40. Reckoning from the occurrence of the rigors, on the 16th, our patient must have been, at the earliest, in the fifth day of the fever,—it might be the seventh or eighth. The description I have just read to you portrays, very accurately, the condition and the symptoms of a patient laboring under the most common form of continued fever now met with in London and our other great towns, about that period of the disease,—that is, not earlier than the fifth day. Now, from this time, the symptoms usually continue of much the same character, with more or less of exacerbation, till the 17th or 18th, or to the 21st or even the 28th day: the most important being those referable to the nervous system,—coma or delirium; to the lungs, congestion, or even pneumonia and pleurisy, which are less frequent; and to the bowels, the diarrhœa.

When a case is about to terminate favorably, these symptoms gradually give way;—the pulse exhibits no tendency to quicken, but rather to fall in frequency; the bowel affection appears easily controllable; the tongue begins to clean at the tip and edges; the patient becomes less stupid; the comatose or delirious state diminishes; the pulse improves in quality, and the general powers of the patient experience a gradual but manifest change for the better. These changes commence generally in or about the third week.

But if the case is not about to end favorably, we shall find an aggravation of some of these symptoms about this period. The pulse will increase in frequency, and its power will be much diminished; the delirium and other head symptoms will become more alarming; or the symptoms referable to the lungs may become more severe,—the breathing more rapid and feeble, and the bronchial tubes impeded by mucus, which the patient has not sufficient power to expel, and, in consequence, death may result from a slow asphyxia; or he may be run down by the constancy or profuseness of the diarrhœa, and perhaps by hemorrhage from the bowels.

Now let us see what was the further course of the symptoms in John Gavin's case.

On the 23d of January he had in some degree recovered the exhaustion caused by his removal to the hospital. His pulse had fallen to 112, but the respirations continued at 48. He was purged four times in the day; the chest signs remained the same.

An enema of starch and opium was ordered at night to counteract the diarrhœa, and his brandy, ammonia, and beef tea were continued as before. The motions became less frequent, and he remained without any change till the 27th.

On this day we found the bowels with a tendency to be loose again; three motions in the day; abdomen tympanitic; pulse 120, and respirations 52. Many of you will remember, that I pointed out to you on this occasion a good mode of estimating the real power of the pulse in fever and other asthenic states, namely, by causing the patient to sit up in bed, and comparing the condition of the pulse in this semi-erect posture with its state in the horizontal position. It was not accelerated by the change from the horizontal position, but its strength and volume became most strikingly diminished; it became very small, and much more compressible, but immediately he returned to the horizontal position, it recovered itself.

There cannot, I apprehend, be a more palpable or unequivocal sign of an enfeebled circulation, than this marked deterioration in the quality of the pulse, on the patient's assuming the semi-erect from the horizontal posture. It indicates very clearly how dangerous it is to remove patients in fever, or other low diseases, from one place to another, or to allow them to move themselves, and how necessary for them it is that they should be constantly attended upon, that every, even the slightest, exertion on their parts should be prevented as much as possible.

It was now evident, that what we had chiefly to deal with was the extreme debility, and the looseness of the bowels. The state of debility was the more fearful, inasmuch as it had come on notwithstanding the free use of stimulants; for since the 21st he had been taking brandy, at the rate of half an ounce every hour. I now doubled the quantity of brandy, and ordered the ammonia and chloric ether to be taken in an ounce and a half of infusion of rhatany every fourth hour.

For the two days (28th and 29th) following this increase of the stimulants, he continued much in *statu quo*,—the pulse 120; respirations 50; the purging diminished, so that he had only one stool in twenty-four hours. The rhonchus in the chest had increased, however, and the heart-sounds were very feeble, so that I felt it needful to increase the stimulant to five drachms every half hour, or thirty ounces in the day.

On the 30th there was some improvement: he was more conscious; the breathing was more free, although still rapid, 50; the rhonchus somewhat less, but the pulse was still 120; he had one loose stool, and the belly was soft.

On the 31st, a still more sensible improvement had taken place. He was much more conscious; the rhonchus was less; vesicular breathing became much more distinctly audible in the lungs; the pulse had fallen to 112, and beat at this rate in the semi-erect as well as in the horizontal posture, although in the former it became reduced in power; the respirations were 46, and the heart's action stronger. No movement of the bowels.

On the following day, the 1st of February, the pulse was down to 100; respirations 45. The tongue was evidently cleaning; the heart's action was stronger; he coughed a good deal, and was rather drowsy. One loose stool.

On the 2d, matters were much the same; pulse 100.

On the 3d, a much more decided improvement had taken ce than had yet been observed. The pulse was only 84, and respirations 38; he was more conscious; the rhonchus was less, and he breathed more freely; the tongue was clean, the abdomen soft, and the bowels quiet.

So far, then, we were in excellent spirits respecting our patient. All the most important symptoms had improved under the high degree of stimulation to which he was subjected; and of these improvements, none was more important than the reduction of the pulse in frequency at the same time that it acquired more power. The least change for the better was found in his consciousness; although he took more notice than before, and was less deaf, and answered questions more readily, he was still very heavy and stupid.

The continuance of this state of stupor led me, on the 3d, to reduce the quantity of his stimulants by six ounces, so that he now took an ounce every hour instead of ten drachms. The chloric ether was omitted. From this time, I regret to say, "a downward tendency," to borrow a mercantile phrase, became evident; the crepitation in the lungs increased, and he began to expectorate a large quantity of thick purulent fluid; his stupor did not diminish; and the pulse and respirations became each day more rapid than the previous one.

On the 4th, the pulse was 116; the respirations 46. On the 5th, pulse 120; respirations 52. On the 6th, pulse 138; respirations 52. On the 7th, pulse 140; respirations 52. And these changes took place, notwithstanding that the largest quantity of stimulants was again administered, and that the infusion of serpentary was substituted for rhatany, with increased quantities of ammonia and chloric ether.

On the 7th, a very serious symptom showed itself, which in part explained the rapid declension of his powers. This was hemorrhage from the bowels. He passed on that day a large quantity of blood by stool, which evidently exhausted him very much. Turpentine was now administered in small and frequent doses, but on the 8th he passed some more blood. He was now evidently sinking, with an extremely rapid pulse and very quick breathing, and he died on the morning of the 9th, which must have been the thirtieth day of the fever.

Here, then, was a case in which no pains were spared to save life, so far as diligent treatment and careful nursing could accomplish that object; it terminated, however, unsuccessfully, and the patient died evidently in a state of extreme

The treatment consisted in the early and free administration of support and stimulants, and in the use of counter-irritation over the chest and abdomen; turpentine stupes were used daily to the front and back of the chest for some time, and afterwards large blisters were applied, and the abdomen was occasionally stuped with turpentine. Close attention was paid to the state of the bowels; astringents were given constantly; and, on one occasion, when the diarrhœa appeared most threatening, an opiate enema was administered; thus the tendency to looseness of the bowels was kept so completely under control, that his weak state could not have been attributed to this. He was supported by a full allowance of strong beef tea, besides milk and arrow-root, and stimulants were given in large quantity, as I have already described.

Now, it behooves us to inquire, why did this patient die? Was there here the *nimia medici diligentia?* Were the quantities of food and stimulants too much for him? Was there any other treatment which we did not use, but which we ought to have had recourse to? Or did death result from causes clearly beyond the control of all medical interference?

The *post-mortem* inspection showed that the morbid changes were limited to the chest and abdomen. In the former there was congestion of the lungs; but to an extent decidedly less than we had expected. The bronchial tubes, however, contained a considerable quantity of the thick yellow purulent matter which he was expectorating during the last few days of his life.

But the most serious lesion was in the intestines. The lower part of the ileum contained numerous deep ulcers, some of which had eaten through the coats of the intestine so as almost to perforate. These ulcers were placed on the free margin of the intestine, and occupied the position of Peyer's patches. In the lower three feet of the ileum, we counted as many as seventeen ulcers, some of which were larger than a shilling. The floors of some consisted only of peritoneum and a little lymph. One very large ulcer existed on the ileac side of the ileo-cæcal valve. In addition, several of the solitary glands were enlarged, and some ulcerated, and the mesenteric glands were enlarged.

I need hardly say, that from our experience of cases of this
and from the diarrhœa, controllable although it was, and
nitis, and the hemorrhage ultimately, we were quite

prepared to find ulcerative disease in the intestine; although, owing to the mildness of the symptoms referable to the bowels, we might well be surprised to find such large ulcers, and so many of them.

This extensive lesion of the mucous membrane of a part of the intestinal canal so important to nutrition as the ileum, must have contributed mainly to the state of prostration of this patient, which persisted for so long a time, notwithstanding the abundant supplies of nourishment which were given him. And yet it is difficult to explain precisely how these ulcerations could have occasioned all this debility, inasmuch as there was no excessive diarrhœa, no great drain from his system, nor did they interfere with the due digestion and absorption of his food, for the quantity of the fæces formed was not unusually great, nor out of proportion to the amount of food taken. It is plain enough, that notwithstanding the disease in the ileum, gastric and duodenal digestion, and chylous absorption in the jejunum, must have gone on sufficiently to admit of the appropriation of the greatest part of the food given.

It cannot, then, be said, that this patient had too much food; if he had, surely we should have found in the bowels large quantities of fæces and portions of undigested food, and during life there undoubtedly would have been flatulence and distress, referable to the stomach, and other signs of indigestion, none of which existed. Nor can it be said that he had too much stimulant; for we had this most striking fact, that with the increase of stimulants the pulse on successive days fell from 120 to 84, and that with their diminution it rose again to 120 and 130. Under the highest stimulation, all the symptoms improved; the chest became more free, the head clearer, the fever less, the tongue cleaner. It was quite evident that both the food and brandy were fully digested and absorbed. We cannot, therefore, plead guilty to the charge of *nimia medici diligentia.* And, on the other hand, I am not aware that anything else could have been done for him besides that which was done. I know of no medicine or remedy more applicable to his symptoms and morbid condition than those which we used. There are those who place great confidence in the powers of mercury to promote the healing of such ulcers as this man had in his ileum. I confess my faith does not carry me so far; and I think most practical men nowadays would eschew the use of mercury, where they had reason to believe that the small intestine was ulcerated, or likely to become so.

The rapid change for the worse which followed the hemorrhage from the bowels, indicated sufficiently that it was the *immediate* cause of death. If the hemorrhage had not taken place there can be no doubt that his life might have been prolonged a few days. But the small quantity of blood lost was quite insufficient to cause death, if there had not previously existed a state of great depression. I have frequently seen much more blood passed by patients who have afterwards perfectly recovered.

I repeat, that were it not for our experience of the constant accompaniment of a state of prostration with a few ulcers of the small intestine, it would be impossible to believe that so grave an effect would follow such a cause. It is true that in this patient the ulcers were not few, but they were found in but a small portion of the intestine, namely, in a space three feet in length, leaving twenty-seven feet of the highest part of the bowel intact. I have, however, seen a state of as great, if not greater prostration, where there were not more than four ulcers. What seems most essential to the production of this state of prostration is, that the sloughing and ulcerative process should be quick, and that it should be perforative in its tendency; that is, that it should eat quickly through the tunics of the bowel, as was the case with Gavin, in whom we found, that at several points the coats of the bowel had been so eaten through as to leave only a little lymph and a thin film of peritoneum as their floors.

But the ulcers are not the only mischief existing in connection with the bowels in these cases: the mesenteric glands are likewise diseased, swollen, and evidently irritated by some abnormal matter passing through them. No doubt the state of these glands interferes with due chylous absorption, but still scarcely sufficiently so to account for the prostration, for the food is freely absorbed in the upper portion of the bowel, and a good deal of it is of a nature (as the oily matter of milk), which must assume the state of chyle, before it can be absorbed.

It seems to me that the production of this state is due not so much to imperfect appropriation of food, as to the absorption of a matter from the ulcerated surfaces, which, circulating with the blood, exercises a poisonous and depressing influence on the system: a matter of the nature of, if not identical with pus, which is absorbed by the lacteals, and perhaps also by the blood-vessels, but probably chiefly by the former, by which route it quickly reaches the lungs, without passing through the liver, where it may contribute to the increase of the bronchial congestion and irritation which so constantly accompany this typhoid state. This view I have often broached to you already at the bedside of patients suffering in this way.

I show you here a preparation which was put up for me some time ago by Dr. Beale.[1] It exhibits a few well-marked deeply-perforating ulcers of the ileum, having much the appearance, from the thick, swollen, and red margins, that the process of sloughing and ulceration was a quick one. In this case (the patient was a young woman), the fever ran its course in about three weeks, the diarrhœa was almost none, and the chief symptoms were a tympanitic abdomen, stupor (in fact coma), bronchial congestion, and extreme prostration. A short time ago, you may remember a woman of the name of Lock, who went off very quickly likewise with similar symptoms, the stupor being so great that I was afraid a few drops of laudanum, administered with starch to check diarrhœa, had narcotized her. There was in this case, in addition to the stupor, bronchial congestion and prostration, but the diarrhœa was very slight, and readily controllable.

Now, that the absorption of pus is capable of producing these depressing effects, we have many proofs.

First, in puerperal fever. In some cases the absorption seems to take place rapidly, and in large quantity; and, under such circumstances, the patient succumbs in a few hours, from rapid prostration and pulmonary congestion, with more or less stupor. In other cases, the absorption seems more gradual, the typhoid condition is induced more slowly but very completely, and, after a time, purulent deposits are found in the joints and muscles, or elsewhere.

Secondly, in cases of erysipelas, in which the suppurative process is rapid, we have typhoid and comatose symptoms, which are out of proportion to the extent of lesion; in such cases doubtless pus finds admission into the circulation.

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[1] The patient's name was Ada Dacon, and the particulars of the case will be found in the next lecture, Case xxviii.

Thirdly, we sometimes have unequivocal evidence of the absorption of pus, as well as to the source whence it comes as with respect to the secondary deposits. I remember attending a case in private practice, where the pus showed itself in the anterior chamber of the eye. This case presented all the symptoms of typhus fever; and for a day or two I viewed it as such. One day I was much surprised at finding pus in the anterior chamber, which increased in quantity very rapidly, and pus was afterwards found in the elbow and shoulder joints. When we came to examine this patient, we found an ulcer in the heart, at the base of one of the mitral valves. Some years ago, we had a case in the hospital of a woman who was suffering from chronic bronchitis; she suddenly became typhoid, and I looked upon it as a case of most aggravated character. She died in a few days, and we found an abscess in the septum of the heart, which had burst, and thus the pus had entered the very fountain of the circulation, producing symptoms nearly resembling those which come on in a case of low typhoid fever.

There seem, then, sufficient grounds for explaining the prostration and fatal termination in Gavin's case, without ascribing any ill effects to either what had been done for him, or to what had been left undone. The sloughing and ulcerative process undoubtedly interferes, to a certain extent, with the function of the bowels, but it also furnishes a source of formation of a poisonous matter, which we know, by experience of analogous cases, when taken into the system, creates symptoms of the same character as those of these fatal instances of typhoid fever.

There is another mode of termination of these cases of typhoid or enteric fever, for which you should yourselves be prepared, and for which you should prepare the friends of the patient, when you may see sufficient reason to apprehend it: I mean, that by perforation. One of those films of peritoneum, which I have already alluded to as forming the floor of many of the ulcers, gives way, and the contents of the bowel pass into the peritoneal sac. In some cases of long duration, when the patient seems to have struggled, day after day, against the assaults of death, rapid sinking immediately follows the perforation, and, indeed, signalizes its occurrence. No new pain is felt, but the patient grows rapidly weaker; the pulse, too, fails, becomes rapid and fluttering, and death from exhaustion or fainting quickly ensues. In other cases, the occurrence of the perforation is ushered in by severe pain in the abdomen; sometimes vomiting; tenderness and pain on pressure; tympanitis; with also increased prostration: all signs of peritonitis, induced by the irritating influence of the intestinal contents upon the peritoneum. When these latter symptoms make their appearance, the free exhibition of opium, in large and frequently repeated doses, is the only measure to which the practitioner can have recourse with any hope of success.

The following case will illustrate the last-mentioned mode of termination, although the direct evidence of perforation, by a post-mortem examination, was not obtainable.

CASE XVII. (Vol. xxxvi, p. 97.) Robert Neek applied as an out-patient at King's College Hospital on the 24th of July, 1852. He was then very weak; his tongue tremulous, coated with a brown fur, and deeply fissured; he was also suffering from diarrhœa. He persisted in following his occupation as long as he could, and was therefore not admitted as an in-patient until the 31st; he was then deaf, and completely prostrate. Brandy and beef tea were ordered, and some days later chloric ether and rhatany.

There was no great change in his condition for about a week; his pulse continued high, his bowels more or less relaxed, rhonchus and sibilus were heard over the chest, and on the 5th and 6th there was slight delirium.

On the 8th there was a considerable fall in the frequency of the pulse; and the record of the 9th is as follows: "Tongue much cleaner; he feels altogether better; the cough has quite left him; the bowels are no longer relaxed."

On the 12th he was still doing very well, and was ordered a slice of mutton. He continued to make favorable progress until the night of the 18th, when he complained of severe abdominal pain. On the 19th all the symptoms of peritonitis were present,—he was lying with his legs drawn up, and the whole abdomen was exquisitely painful on pressure. A grain of opium was given, and ordered to be repeated.

He passed a delirious, restless night, the same symptoms continuing, and died on the 21st.

Had our patient Gavin not been carried off by the exhaustion consequent on hemorrhage and purulent infection, it is very probable, from the state of the ulcers, that perforation must have taken place, of which he would have died in either of the two ways which I have described.

A third mode of termination is by colliquative diarrhœa. The patient may be going on well, and the practitioner may even be sanguine in his expectations of a favorable result, when the diarrhœa may suddenly become colliquative, and a few discharges of large watery evacuations will terminate the case.

But to return to the treatment of the patient Gavin. It may be said, surely the irritation of the bowels was kept up by all the stimulants (to say nothing of the food) which were given, and had they been more sparingly supplied, the ulcerative process in the ileum would not have gone so far.

This notion respecting the injurious effects of alcoholic stimulants, in cases where there is a tendency to bowel affection, is, I think, partly founded upon a vague supposition that the alcoholic fluid comes in direct contact with the irritable mucous membrane. Now the reply to this is, that we have the strongest reason to conclude that fluids of this kind never, except when taken in very large quantity at one time, pass the pylorus, but are absorbed by the walls of the stomach. This is especially the case when they are administered in the way I recommend,—that in which they were given in Gavin's case,—namely, in small quantities, with intervals of not less than half an hour between each dose. Thus one dose is absorbed before the other is given.

But it may be urged, that the alcohol gets into the blood, circulates with it, and so increases the tendency to ulceration.

Upon this point we can only appeal to experience. The administration of alcohol to healthy persons does not prove injurious by any irritative effects it may produce on the bowels. Of all the ill consequences which the advocates of the teetotal system, in their most praiseworthy zeal, have summed up as likely to be caused by the use of alcohol, I do not find that diarrhœa or ulceration of the bowels is noticed; and were it a frequent effect, it certainly would not have escaped the scrutiny of these gentlemen. It is true that a debauch, in which a man may drink at one sitting as much, or considerably more than we should think of giving in twenty-four hours, may sometimes disturb the liver, and, through its increased secretion of bile, the bowels; but the looseness thus excited seldom or never proves otherwise than salutary.

Nor do we find that effects of this kind are apt to follow the liberal administration of alcoholic stimulants in other low diseases; in erysipelas; in the diffuse inflammation of the areolar tissue, whether traumatic or not; in puerperal cases; and we give it repeatedly in cases with threatened or actual ulceration of the bowels, without any increase, but, on the contrary, a marked diminution of the unfavorable symptoms. Such, indeed, was the case with our patient Gavin. On the first few days of his taking stimulants, a manifest improvement took place in all his symptoms, those affecting the bowels as well; so much so that until the post-mortem examination revealed the true state of matters, I blamed myself for diminishing his supply of stimulants on the third. Probably the good effects continued until the puriform matter had entered the circulation in sufficient quantity to produce its poisonous effects.

I could enumerate many instances in which this mode of treatment, by free stimulation, was of great and signal advantage. But I must content myself with mentioning a few, referring particularly to some cases of this kind which have lately been treated in the hospital.

CASE XVIII.[1] (Vol. xxxi, A. p. 54.) Many of you will recollect the case of Lucy Wood, aged 14, who was in the house about three months ago. She took as much as an ounce and a half of brandy every hour for three days together, and for the next fortnight half an ounce was hourly administered; this latter quantity, however, being sometimes much increased as occasion required. Under this large amount of stimulants, her symptoms gradually improved, and she was discharged quite well on the 4th of December, having been about nine weeks under treatment.

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[1] From the notes of my clinical clerk, Mr. Simpson.

This girl labored under the great disadvantage of heart disease. A loud systolic bellows sound, heard most distinctly at the apex of the heart, was present when she came in, and resulted from an attack of rheumatic endocarditis, which occurred some time ago. She was admitted on September 26th, and on October the 4th her symptoms began to assume a very severe character, and she was evidently getting very low. At this time she was also suffering from diarrhœa, for which she was taking astringents with chloric ether, and on one occasion it was thought advisable to administer an opiate enema.

On November 1st, she was in a state to warrant us in diminishing the quantity of stimulants. The pulse had now fallen to 120; on the 4th it was 114. After she had recovered from the fever, a very painful node formed upon the anterior surface of the tibia, which ultimately did perfectly well.

CASE XIX.[1] (Vol. xxxi, A. p. 57.) John Bigg, æt. 15, was admitted with fever, on the 3d of September, 1850. The attack had begun with shivering and the other usual symptoms five days before. He soon became delirious. A stimulating plan of treatment was commenced at once.

On the next day, the 4th, his nose bled, and looseness of the bowels came on; the urine contained a little albumen; the pulse numbered 112. Chloric ether and krameria were given, and in the evening the wine was increased to between five and six drachms every hour.

On the 5th, rose spots were developed; the purging continued, and about a pint of blood was passed at stool. Enemata of starch and opium were administered, and ten-minim doses of turpentine were given every fourth hour. In the evening brandy was substituted for wine. There was then great prostration, muttering delirium, and cold feet; and as he had not slept since admission, ten grains of Dover's powder were given. The next day his pulse was 150. All the same symptoms continued, and there was still blood in his stools. During the 8th, 9th, and 10th, the symptoms were the same, but less severe. He complained, however, of great abdominal pain and tenderness, for which turpentine stupes were repeatedly applied, and the brandy increased to an ounce every hour. The albumen disappeared from the urine.

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[1] Reported by Mr. Simpson.

On the 11th, a little blood again appeared in the motions.

On the 12th, there was a slight sweating, apparently critical. On the 13th, he was much better, and from that day continued to improve. The brandy was reduced gradually, and on the 24th he was convalescent and on full diet.

Case XX.[1] (Vol. xxxi, A. p, 67.) Elizabeth Bevan, thirty-six years of age, an overworked needle-woman, was attacked with the ordinary premonitory symptoms of fever, September 15th, 1850, and admitted in a semi-conscious state four days after. Half an ounce of brandy was given with strong beef tea every hour, as well as chloric ether and ammonia; and turpentine fomentations were applied to her belly and chest.

The next day she lost about a pint of blood by stool, and became very pale and low. Pulse 122; respiration 33. Turpentine and krameria were administered, and enemata of starch and opium; the brandy also was increased to a drachm and a half every quarter of an hour, with beef tea of three times the usual strength.

On the 19th there was little change: she was still purged, and vomited repeatedly, the latter symptom obliging us to administer the stimulants only in still smaller quantities at a time and more frequently. Enemata of beef tea, quinia, starch, and opium were given, and afterwards of tannic acid and turpentine. Her pulse continued about 120.

On the 20th, the diarrhœa abated: there remained however cough, difficult respiration, 34, and expectoration of tenacious mucus, while catarrhal sounds were heard over the chest.

From this time she began to mend, although slowly; the brandy was reduced, but the pulse continued very quick for more than a week. On the 29th she was reported as "improving generally," and on the 4th of October, as "rapidly recovering;" she was then on full diet, and taking no medicine.

On October 25th she was discharged cured.

Case XXI. (Vol. xxxi, B. p. 162.) Charles Perugia, æt. 20, admitted July 3d, 1850. His illness began with shivering, followed by fever, about a week before admission, and the last three or four days he had suffered from purging. He was ordered half an ounce of wine every two hours, beef tea, and chloric ether in decoction of logwood.

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[1] Reported by Mr. Si

On the 4th, the fever continued; his tongue was half protruded, tremulous, and brown; he was restless, weak, and took little notice; a few small rose spots were apparent. Pulse 96. The diarrhœa had ceased. His head was shaved, and a third of an ounce of brandy given every hour.

On the 6th severe purging returned; and on the 8th he passed a good deal of blood in his motions; these symptoms were checked with difficulty by enemata of half a drachm of tannic acid, half a drachm of laudanum, and starch. Pills containing tannic acid and quinia were also given.

By the 12th he was much better; his pulse 80; the motions natural. By the 15th, his appetite and hearing had returned; and by the 20th he was up and convalescent.

All these may be called desperate cases, in which the pulse was very rapid and feeble, and the tendency to death from exhaustion very great. All were accompanied by diarrhœa and hemorrhage, which in all became considerably less under astringents and the largest doses of stimulants. A general disposition to hemorrhage seems, in fact, to be a consequence of the deterioration, perhaps of the disorganization, of the blood by the typhoid poison; and this is manifested not only in the passage of blood by stools, which might otherwise be considered simply as a direct result of ulceration, but by hemorrhage from the nose and kidneys, by the presence of petechial spots in the skin, and occasionally, by extravasations of blood elsewhere.

Associated, and possibly connected with this disposition to hemorrhage, we sometimes find, after death, a remarkable condition of the spleen—that organ being softened, sometimes pulpy, and breaking down readily under pressure with the fingers.

The case of Charles Andrews (Case xxxi), which I shall give in detail in my next lecture, and the two following fatal cases, will illustrate these remarks, and the extent to which the hemorrhagic tendency is sometimes developed.

CASE XXII.[1] (Vol. xli, p. 15.) Sarah Ann Chandler, a widow, 39 years of age, was admitted, May 10th, 1853, with symptoms of fever. Five days before she had been much shocked by suddenly hearing of the death of her father, and to this she attributed the commencement of her illness: she said that she felt at first as though she had received a heavy blow.

On the 11th her face looked flushed and anxious; her lips were dry and soiled; her tongue covered with a thick white crust; the conjunctivæ injected, and her eyes somewhat suffused. There was cough and abundant frothy expectoration, with sharp shooting pain in her chest, and rhonchus and sibilus with some crepitation were heard. Pulse 136; respirations 36. Carbonate of ammonia and chloric ether were given. At night she passed three or four evacuations; and the next day her pulse rose to 180, and the respirations to 42.

On the 13th she seemed worse; her pulse was very feeble and thrilling to the finger, and had fallen to 84. The respiration was more embarrassed, and, on auscultation, large crepitation could be heard all over the chest. She had ceased to cough and expectorate,—apparently from want of power.

On the 14th she appeared sinking; she could hardly speak, and her pulse was scarcely perceptible. A blister was ordered. She died in the evening.

At the post-mortem inspection, the lungs were found slightly congested, and the tubes much choked with secretion, but otherwise healthy.

The heart was flabby and somewhat soft.

Petechial extravasations of blood were found among some of the muscles of the chest, more or less symmetrically situated on either side.

Neither Peyer's patches nor the solitary glands were found diseased.

*The spleen* was a good deal softened, presenting much the same appearance as in the patient Charles Andrews (Case xxxi).

CASE XXIII.[2] (Vol. xlvii, p. 15.) George Rose was admitted

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[1] From the notes of my clinical clerk, Mr. (now Dr.) Plowman.

[2] Reported by Mr. Hardwich.

May 10th, pale, exhausted, and only partially conscious, but with a rapid, feeble pulse, quick respiration, and hot skin. He had felt languid and weak for a fortnight, but had given up work only five days. Blood had been passed both by stool and urine.

During the short time he survived his admission, two motions were passed approaching to a pitchy blackness, and one of a blood-red color, and also bloody urine. He was restless, moaning, and delirious. Towards evening, the exhaustion increased rapidly, and he died early on the morning of the 11th.

At the post-mortem examination, the patches of Peyer were found ulcerated, especially at the lowest part of the ileum. Many of them were covered with a red fungous mass; others simply enlarged and injected.

I have felt it a duty to make these remarks to you upon the subject of the treatment of fever by stimulants (and they apply no less to the treatment of other exhausting diseases,—erysipelas, influenza, bronchitis, carbuncle, &c.), because I wish to caution you against the morbid fear of over-stimulation, which leads many to adopt an opposite or a vacillating course, and to allow their patients to die from exhaustion. This is the mode of death to which fever patients are peculiarly prone; and I hold that the lower you allow them to become at first, the more likely is the ulcerative process in the intestines to take head, just as it is apt to do in the bowels and in the corneæ of the eyes, in cases where there is an insufficient supply of properly nutritious food. At the same time, I must beg that you will not run away with the notion, that every patient in fever, about whom you may be consulted, is to be treated with thirty ounces of brandy a day. There are many cases in which no stimulant at all is necessary; others, again, in which it is not needful to give more than four or six ounces a day. You must bear in mind that we have two classes of cases of fever to deal with, the mild and severe; or those which have had a large, and those which have had a small dose of the peculiar poison on which the febrile state depends. Where a large dose of the poison has been received into the system, you will generally find it necessary to give large quantities of alcoholic food, or the patient will not have sufficient vital power to resist its depressing effects. Some few instances, indeed, there are, in which the dose of the poison is so large, that the patient never rallies from the state of almost complete paralysis which it induces; such cases run their course in twenty-four or forty-eight hours, or within a week. The case of S. A. Chandler (p. 90) was of this kind. But the mild cases,—and fortunately, in many epidemics, these are the most numerous,—do perfectly well on a very moderate amount of nourishment, with little or no alcohol.

The objections which some excellent practitioners have to the use of stimulants, apply with more justice to the slovenly mode in which they are too often given. Generally left altogether to the discretion of a nurse, they are given in large doses at one time, or with other food, or without reference to the medicines which are being likewise administered; they consequently create derangement of the primary or stomach digestion, flatulence, and flushing. If you give alcohol, give it with due regard to its digestion by the stomach, and so as not to interfere with the other food or the medicines likewise being taken.

I am convinced that it is much better to err on the side of over-stimulation than not to give enough; for if we have over-stimulated a patient, it is very easy to pull him down again; there are plenty of appliances and means for this purpose; but if the patient sink too low, nothing is more difficult than to restore him. If by your feeding and stimulating, the thermometer of life has risen to too high a point, nothing is easier than to depress it; but if fallen below a certain point, then to raise it again, much more to restore it to the height from which it fell, *hic labor, hoc opus est.*

In conclusion, let me say a word or two as to the treatment to be pursued, when you have reason to fear that the bowels are ulcerated. It seems to me, that the great principle of treatment in such cases, is to keep down peristaltic action, which is best done by opium and astringents containing tannic or gallic acid. Many attach great value to the use of sulphate of copper; but as it is generally given with opium, and does not always agree without opium, I think the latter drug has the largest claim to the good services often done by the combination. When hemorrhage occurs, nothing is so effectual to restrain it as turpentine given in small doses, so as not to risk offending the stomach;

even so small a dose as five minims is often sufficient; and I frequently apply it externally, as a stupe to the walls of the abdomen, with decided benefit. In dealing with these cases, you must not be timid as to allowing the bowels to remain inactive for even several days. I have never seen any bad consequence from their not acting even for four or six days; and when they are to be provoked to act, let that be done by some simple enema rather than by aperient medicine.

# LECTURE V.

## *On Continued Fever.*

In offering to you to-day some remarks on those forms of continued fever which are most likely to come before you, let me first say a word or two respecting the intimate nature of the most prominent clinical feature of the disease before us, from which, indeed, it derives its name,—I mean *fever;* for it is of great practical importance to have something like definite views upon this point of pathology.

You all know that *fever* is marked by a hot, sometimes burning, often flushed, and generally dry skin; by a quickened pulse, loss of appetite, thirst, accelerated respiration, and more or less loaded urine; and these phenomena soon become accompanied by a manifest wasting of substance and loss of power.

We can best explain these symptoms by supposing that a poison, circulating in the system, interferes with, and greatly modifies the processes of nutrition and secretion; what Dr. Prout has called the *secondary destructive assimilation* is exalted, and the elements of the tissues seem to undergo a rapid oxidation. The result is a rise in temperature throughout the systemic capillary circulation, generally wasting, and more or less rapid exhaustion of vital power.

Now symptoms of this kind always follow the introduction of a poison into the system, and are indicative of a peculiar disturbance, which the presence of that foreign matter in the blood establishes. You have every day the experiment performed millions of times, of introducing into the blood a minute quantity of vaccine lymph, through a puncture in the arm. This, in a few days, establishes a definite form of fever, with certain local phenomena in the shape of one or more pustules at the seat of the wound. So if the analogous poison of small-pox gets into the system, as we call it, or more correctly into the blood, a definite fever is established, with the local development of peculiar pustules on the skin and mucous membrane. The same may be said of all the fevers which we call *exanthemata.* In each there is a definite poison, and that poison produces definite febrile phenomena. If left to itself, the fever begins on a certain day and ends on a certain day, and affects the skin or mucous membrane, either gastro-intestinal or respiratory, or even genito-urinary, in a specific manner. Take, for instance, the poison of scarlatina: it quickly establishes an intense fever; it attacks the mucous membrane of the throat and that of the kidneys; it develops a peculiar rash on the skin, and more or less of irritation and swelling of the cervical glands. In such cases, although the great intensity of the fever is in the early stages, it nevertheless continues more or less in a chronic form until all local phenomena have disappeared.[1]

What the pathological significance of the local phenomena of fever may be, we are scarcely yet in a position to declare; but it seems very probable, that they have at least much to do with the process of elimination of the poison. Each poison has apparently an elective affinity for some particular structure or organ, and through it makes for itself a channel of escape out of the system. Thus it seems very reasonable to suppose that the cutaneous desquamation, which so often occurs in scarlet fever, is one medium for the extrication of at least a part of the poison from the blood. In like manner, the pustules of smallpox are, in all probability, due to a nisus of elimination, and each pustule is a point of exit of a certain quantity of poisonous matter.

When there is so great a diversity of symptoms, it is not too much to suppose that the poisons, upon which these various forms of fever depend, are also essentially different from each other. There are, in fact, as many poisons as there are fevers. And the greatest number agree in this, that they give rise to a febrile state which is continuous, or, according to the medical term, *continued,* until it exhausts the power of the patient, or if his strength will permit, until the poison is eliminated.

One poison is distinguished by the extraordinary peculiarity, that, after infecting the system for a certain time, giving faint or no indications of disturbance, a form of fever is engendered which is distinguished by more or less complete remissions of the febrile state. These occur periodically, and form part of a peculiar train of phenomena, consisting of a cold or shivering state, a hot febrile state, and a sweating state or stage, out of which the patient gradually passes into a non-febrile state, or one of apyrexia, and remains quite well until an interval of twenty-four, or forty-eight, or seventy-two hours has passed by, when the same train of phenomena will be repeated.

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[1] Professor Parkes' invaluable "Lumleian Lectures on Pyrexia" deserve careful study by all who take an interest in the pathological phenomena of fever.—*Medical Times and Gazette,* March 17th, 1855.

It would almost seem as if the marsh or paludal poison, upon which this fever depends, underwent, with varying rapidity, some increased development, at the acme of which the peculiar three-stage phenomena come on; these subside with the elimination of a certain portion of the poison from the system by the sweating process, to be renewed when in due course a fresh development of the poison takes place.

It seldom happens that the marsh poison, once admitted into the human system, ever becomes perfectly eliminated from it; and persons once infected, are for this reason ever after liable to renewed attacks, under even the slightest malarious influences.

When that curious compound pus, a product of disintegrated tissue, enters the current of the circulation, it engenders a peculiar fever, of which the phenomena are increased heat of skin, accelerated pulse and respiration, and depression of nervous power, sometimes so great as to kill very quickly by sheer exhaustion. But, in most cases, the fever persists, and soon signs of elimination show themselves in local collections of pus in various parts of the body. After these have been evacuated, if the vital powers of the patient are sufficient to bear up against the trying and exhausting process, often of tedious duration, recovery takes place.

I must limit my remarks on this occasion to the subject of *continued fever.* Of this, it may now be fairly admitted that there are three varieties, as proved by the excellent researches of Stewart, Jenner, and others in this country, America, and on the Continent. These are the Typhoid, Typhus, and Relapsing fever, each produced by a distinct, although doubtless very similar poison. As the Relapsing fever is comparatively of rare occurrence, I shall confine myself to the *Typhoid* and *Typhus* varieties.

The term *Typhoid* is applied to that kind of continued fever which is accompanied by catarrh, diarrhœa, or a tendency to it, and more or less abdominal tenderness and tympanitis; and in which, after death, we find a morbid condition of the solitary glands, and of Peyer's patches in the ileum, amounting sometimes to irritation and enlargement only, in other cases to sloughing and ulceration. Another feature, characteristic of typhoid fever, is the development of a peculiar eruption of circular, slightly elevated, rose-colored spots, often of considerable size, which fade or vanish momentarily under pressure; these make their appearance from the fifth to the twelfth day, or even later, and are generally confined to the chest, belly, or back.

The appellation *Typhus*, on the other hand, is applied to those cases in which the symptoms of intestinal irritation are absent, and which are marked by a copious eruption, consisting of small, irregular, reddish, or purplish spots, which generally run together so as to form irregular or crescentic patches, not confined to the chest and abdomen, but often to be found on the extremities, and indeed covering almost the entire surface, and on the whole nearly resembling the eruption of measles.

The case of Gavin (Case XVI), which I detailed to you in my last lecture, is a good example of the Typhoid form of fever, excepting that the rose spots were not developed. The following case is, in some respects, a better illustration, as the catarrhal symptoms, the rose rash, and the diarrhœa were all well marked; it is also a good example of the treatment which I advocate in these cases.

CASE XXIV.[1] (Vol. xxxix, p. 143.) M. A. Copstock, a nursemaid, eighteen years of age, was admitted into King's College Hospital, February 5th, 1852. Her illness commenced eight days before, with pains in her limbs, which she attributed to some trifling exposure to cold and night air. Carbonate of ammonia was administered in five-grain doses every third hour.

On the third day after admission, her condition was as follows:—

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[1] Reported by Mr. (now Dr.) Plowman.

Her face looked puffed and heavy, her eyes suffused; she complained of headache, and seemed confused and drowsy; the skin felt hot and dry, and the tongue was coated with a whitish fur. There was a troublesome cough, with expectoration of a scanty viscid mucus of rusty tinge. On listening to the chest, slight rhonchus and crepitation were heard here and there, both in front and behind. There was considerable tenderness on making pressure over the belly, and she had passed three copious liquid evacuations during the night, and two that morning. No spots were then found on the skin. Her pulse was 108, and respirations 38.

An enema of starch and opium was directed to be given after every loose stool, and turpentine stupes to be applied to the belly.

On the next day, the 8th, beyond the check to the diarrhœa by the enemata, there was no distinct alteration, either for worse or better, in the general condition of our patient. Six drachms of brandy were ordered to be given every second hour.

On the 9th, she was extremely drowsy and unwilling to be disturbed. The brandy was increased to an ounce every hour.

On the 10th, the fourteenth day of the disease, all the same symptoms continued, but on the whole she seemed better. A number of scattered rose-colored spots were observed, for the first time, on the chest and belly. She showed a great aversion to the brandy.

There was no material change on the 11th and 12th, but as the diarrhœa continued, the ammonia was given in decoction of logwood, and the starch and opium enemata were administered as before, with decided benefit.

On the evening of the 12th, she seemed more prostrate, and the brandy was increased to an ounce every half hour.

Her condition on the 14th was scarcely better, the same symptoms continued: breathing urgent, 48 times a minute; pulse 116; cough frequent and hard, with expectoration of simple mucus; profuse liquid evacuations recurring from time to time, but kept in check by enemata; the same drowsy condition and dislike to disturbance; the hot and dry skin, with scattered spots becoming fainter. She continued to exhibit the greatest aversion to the brandy and beef tea, swallowing very imperfectly what was put in her mouth, so that the proper amount of food and stimulants were administered with great difficulty. This is not an uncommon feature of such severe fever cases as that of this patient; it demands great firmness on the part of the practitioner, and in no conjuncture will he more require the active co-operation of an experienced nurse; timid and anxious relatives and friends are not to be depended on in such emergencies.

On the 15th, as there was some increase in the catarrhal sounds heard over the front of the chest, turpentine stupes were ordered night and morning. In the evening she became more drowsy; her head was therefore shaved, and acetum cantharidis applied to the scalp.

On the 16th, which was about the twentieth day of the disease, a very decided improvement took place: she was more lively, slept quietly, and a profuse perspiration, in all probability critical, burst forth; her pulse fell to 112, and the respirations to 40. This improvement continued the next day; her tongue and lips began to clean, and the eruption had disappeared from the skin. On visiting the hospital in the afternoon, I found her again more drowsy, and thinking that the drowsiness indicated over-stimulation, I reduced the brandy from an ounce to six drachms every half hour. After this the drowsiness passed off, but for many days the improvement was slow; her cough continued troublesome, the bowels relaxed, and the pulse and breathing high. A second attempt to reduce the stimulants led only to their renewal in the previous doses.

From the 25th (the twenty-eighth day of the fever) the pulse fell in frequency rapidly; the brandy was reduced. It was not, however, until the 4th of March that the cough had given way: she then felt well and anxious to get up; her pulse was 80 and respirations 30; she slept well and her appetite was good. She left the hospital on the 12th (forty-eighth day) quite recovered.

You will not often meet with so severe a case as this ending in recovery. I cannot but believe that the favorable result was owing to the steady exhibition of support of all kinds, especially of stimulants, from the earliest period of the disease. Still it is curious to observe, how about the twentieth day a marked favorable change took place, and was accompanied by a profuse sweating, apparently of a critical nature.

As a good example of the *Typhus* form of fever, allow me to direct your attention to the particulars of a case which proved fatal in the hospital in September, 1853, and which I shall have to refer to again.

CASE XXV.[1] (Vol. xliii, p. 103.) E. Church, a man, aged 59, was attacked with shivering and pains in his limbs. In the course of a few days he was too ill to remain up and about, and therefore took to his bed. He suffered chiefly from great pain in his head, and there was some delirium. He was admitted to the hospital, September 18th, 1853, about a week after the shivering. He was then not sufficiently conscious to understand what was said; his tongue was dry and coated with brownish black sordes; pulse 100; *a measly rash covered the whole front of the chest and abdomen.*

A blister was applied to the scalp, ten minims of chloric ether with five grains of carbonate of ammonia were given every four hours, and half an ounce of brandy with beef tea every half hour. The brandy was increased to six drachms in the afternoon.

He continued delirious through the night, and in the morning seemed more insensible; he passed his urine unconsciously, and could with difficulty be made to take the brandy. There was continual hiccough; pulse 100. A large blister was applied so as to cover the lower part of the chest and the stomach.

On the 20th the delirium rapidly gave place to coma. The hiccough continued. Pulse 92. In the evening the urine was found to be albuminous and to contain blood casts; and on this account the physician's assistant discontinued the stimulants. At ten o'clock his pulse was much weaker, and had risen to 110; at eleven, it was imperceptible. Convulsions and death followed shortly afterwards, the disease having existed only ten days.

On examining the body after death, a small quantity of serum was found under the arachnoid. The lungs were much congested and also the spleen. There was no evidence of kidney disease excepting a slightly granular appearance.

It may be suggested that this was a case of uræmic poisoning from renal disease, and not typhus at all. The measly rash was sufficient evidence of typhus, which in this case may have attacked a subject suffering from diseased kidney in an early stage. No doubt the influence of the poison in this case would embarrass the action of the kidneys more than it is well known to do even when those organs are healthy.

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[1] Reported by Mr. C. Macnamara.

Admitting as I do the existence among cases of continued fever of two clinical varieties, the typhoid and typhus, I am nevertheless convinced that instances every now and then occur, in which the distinction cannot be made, unless the presence or absence of enteric symptoms alone, or of some other *single* symptom, be taken as diagnostic. The following cases will serve to explain my meaning.

CASE XXVI.[1] (Vol. xl, p. 264.) Daniel Ragen, æt. 24. His illness began on the 2d of March, 1854, with headache, but no distinct shivering; and he was admitted on the 6th with fever, a rapid pulse, a brown and dry tongue, and suffering great pain in his head. Severe headache continued throughout, as a prominent feature of the case.

On the evening of the 8th, he was twice purged, and a mixture of chloric ether and decoction of logwood was ordered; also half an ounce of brandy every two hours, and beef tea. Some rose spots were observed thickly scattered over the belly and chest. Pulse, 104, respirations 22.

The next day the purging had ceased, the other symptoms continued, and he coughed and expectorated some brownish mucus. It was thought advisable to shave his head on account of the pain.

On the 10th a measly eruption was fully developed on his chest, belly, and back. A blister was applied to the scalp.

On the 11th, the respiration was rapid and labored, 44 times in a minute; the pulse continued the same; there were some drowsiness and delirium; the diarrhœa returned, but was controlled by an enema of starch and opium.

On the 15th, being the thirteenth day of the attack, the pulse had fallen to 96; there was great improvement in all the symptoms, and the enema and logwood were discontinued. The following day there was some return of diarrhœa, which was checked by a repetition of the enema. He expectorated, with difficulty, a viscid mucus, which seemed to choke up the lungs. General improvement continued; and by the 20th, his pulse had fallen to 76. On the 22d, he was pronounced quite convalescent. He remained some time longer in the hospital, with pain in his side.

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[1] Reported by my clinical clerk, Mr. Bird.

This, then, was a case in which, with a well-developed rubeoloid eruption, enteric symptoms were nevertheless present; it had some features of typhus, and others more prominent of typhoid, and there was the occurrence of the two eruptions in one person.

CASE XXVII.[1] (Vol. xliii, p. 39.) John Cahill, aged 42 years, was attacked June 16, 1853, with pain in his head and hips; but, although ill, he continued his work until the 19th, when he was seized with shivering, and increased pain in his head and limbs, accompanied by total prostration of strength and loss of appetite. He had been purged excessively by a dose of salts. One of his children and several people in the same street were suffering from fever. He was admitted on the 28th.

On the 29th he complained of cough and sore throat; his bowels had been twice moved: a number of deep rose-colored spots were observed covering his body. Pulse 104, respirations 28. He was ordered a mixture of chloric ether and ammonia, to be taken every third hour, and half an ounce of brandy every two hours.

On the 30th the pulse and respirations were 100 and 24 respectively. Bowels open once.

By the 2d of July, the spots had almost died away. Pulse 100, respirations 23. On the 3d, the pulse and respirations were 104 and 24; on the 4th, 112 and 28. The bowels continued regular. On the 5th, the pulse had fallen to 96, the respirations remained 28. He still complained of sore throat, for which a blister was applied over the larynx.

On the 6th, about the twenty-first day of the disease, he was in every respect much better: his appetite good, pulse 90, respirations 24.

From that time he made a rapid recovery. On the 8th, the pulse and respirations were 80 and 20 respectively; on the 10th, 78 and 20; and on the twelfth, 78 and 24. On the 24th, he was discharged well.

Cases of fever are occasionally met with, which run their course and prove fatal, without the occurrence of any serious diarrhœa, and yet, on making a post-mortem examination, extensive ulceration is found in the ileum. I well remember a case of this kind which proved fatal in the hospital as long ago as the beginning of 1850.

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[1] Reported by Mr. C. Macnamara.

Case XXVIII. (Vol. xxviii, p. 32.) The patient's name was Ada Dacon; she was eighteen years of age. Her illness commenced in the ordinary way, with rigors and pain in her head, back, and limbs, and had lasted a fortnight when she first came under treatment, January 19th, 1850. She was then in a high state of fever, and complained of frontal pain, great depression, loss of sleep with frightful dreams; there was some abdominal tenderness, but no relaxation of bowels. Her pulse and breathing were 120 and 30.

On the 22d, some of the aromatic spirit of ammonia was ordered to be given in camphor mixture every six hours, and six ounces of wine in the day.

On the 24th, there were bronchial râles heard pretty extensively; she could not sleep, but lay drowsy and moaning. Pulse 130, respirations 32. A blister was applied to the back of the neck, and a mustard poultice to her chest.

On the 25th, or the 21st day of the fever, she was much sunk; her pulse and breathing were increased in frequency to 144 and 44; she had passed two relaxed motions, for which an astringent and stimulating mixture was ordered. There was no return of diarrhœa, but she continued in much the same state until the 29th, when she became rapidly worse and insensible, and died the following morning.

At the post-mortem examination, we found numerous well-defined ulcers, situated in the lower part of the ileum, and one or two large ones, involving the ileo-cæcal valve.

Besides such well-marked exceptional cases as XXVI and XXVII, we meet with others, from time to time, in which the eruption is not either of the typhus or typhoid kind, but something intermediate; and in which abdominal symptoms may or may not be present. Others again, though well-defined and even fatal cases of continued fever, will exhibit throughout no [illegible] of any kind.[1]

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briefly two other cases to illustrate these exceptions.

admitted with fever, July 11th, 1855 (vol. xlviii, p.

Besides the specific eruptions, we frequently meet with dark, purplish specks of a variable size, called *petechiæ;* they are produced by little extravasations of blood beneath the cuticle, and of course do not disappear on pressure. They are not peculiar to the fevers we are considering, but are common to them and to other fevers and diseases of debility. They were present in the following low typhoid case, and the spots in the patient Selby (Case XXXIV) had very much the petechial character.

CASE XXIX. (Vol. xxxvi, p. 24.) Emma Turner, æt. 17, was admitted a fortnight after shivering, with symptoms of low typhoid fever. Her pulse was then weak and rapid, 128; respirations hurried, 36; her tongue was dry and brown, and her teeth and lips were covered with sordes. She was passing loose, dark, and extremely fœtid motions, and complained of great abdominal tenderness. Petechiæ were present on the abdomen and back. On listening to the chest, rhonchus was heard both in front and behind. She was constantly moaning, occasionally screaming out, and delirious.

Half an ounce of brandy was given every two hours; also chloric ether and astringents, opiate enemata, and turpentine stupes to the belly and chest.

For three days she remained in much the same state: the bowels continued relaxed and the motions were passed unconsciously, the pulse ran high, food and stimulants were administered with great difficulty.

On the fourth day after admission, she sank into a state of stupor, and died at night.

106). He had then been ill about a fortnight. He was deaf, and his body covered with *rather large rose-colored spots.* Pulse 120. *His bowels had been costive and were still confined.* He was treated by moderate stimulation. On the 14th he was sweating freely; pulse 92. He continued improving daily, and by the 20th was pronounced convalescent. There had been no looseness of the bowels throughout.

Joseph Garland, æt. 18 (vol. xlii, p. 85), was attacked with sickness, giddiness, pains in his limbs and bleeding from the nose; and some days after by shivering, followed by severe fever and *loose bowels.* He was admitted on the 19th May, 1854,—the seventh day. His eyes were bloodshot and suffused, his throat sore, his breathing rapid; *an indistinct diffused rash covered his chest and arms.* He continued very ill *and much purged* for five days. Brandy and beef tea were regularly administered, and the diarrhœa restrained by opium enemata. On the 24th there was decided improvement, and by the 29th he was convalescent.

There is yet another form of eruption, which you will have frequent opportunities of seeing in fevers of this class—an eruption of minute, pearly vesicles, scattered in profusion on the skin of the neck, chest, &c.; these have been called *miliaria* or *sudamina*, and, as I mentioned in my lectures on rheumatic fever, are not peculiar to any one disease, but common to many, and indicative generally of a sweating state; hence I prefer the term *sudamina* to *miliaria*. I may add that they do not require a general sweating for their development; a local sweating, such as may occur in a fissure between folds of the skin, is often sufficient to bring them out there.

I must now proceed to consider, briefly, the more common complications of fever: those involving the lungs, brain, bowels, or kidneys, which I have not yet mentioned; and the plan of treatment I usually pursue in each.

Whenever the natural interchange of material between the blood and the tissues is imperfectly performed, the capillary force of the circulation is deficient, and the circulation through the capillaries becomes sluggish and imperfect. This is what occurs when an unhealthy blood is circulated—blood, for example, charged with the poison of typhus, or with urea; and we consequently find, in all such poisoned conditions of the blood, a tendency to local congestions, often of vital organs. Hence it is that we meet with pulmonary congestion as a common complication of continued fever; it is a purely passive congestion due to the altered quality of the blood, and it has no resemblance, except as regards the hyperæmia, to inflammatory congestion. When fairly established and persistent, it is marked by wheezing, and more or less of crepitation, with increased bronchial secretion of mucus, occasionally tinged with blood, which the patient coughs up. This condition was well marked in the patient Copstock (Case XXIV), also in Selby, to whom I have before referred, and whose case I will quote in detail presently (Case XXXIV). The case of Emma Turner (Case XXIX), just referred to, affords a third example.

Instances might undoubtedly be found of the occurrence of a true inflammatory bronchitis, and even of pneumonia, in the course of continued fever; but these must be looked upon as extraordinary complications. I could instance several fatal cases of fever, in which patches of lung have been found carnified after death. In these cases, the congested state of the lungs was intense, and the solidification seemed due to the great engorgement and increased secretion, rather than to plastic exudation. At the same time, this exudation now and then takes place both into the air-cells and on the pleural surface, but I doubt not that it is then simply the result of the mechanical retardation of the blood in the finest bloodvessels.

Case XXX.[1] (Vol. xli, p. 58.) Sarah Beeson, aged twenty-three, an artificial flower maker, was admitted into King's College Hospital, July 9th, 1853. She had been living badly, and in the midst of bad smells, and working hard.

On admission, she was scarcely able to stand; she complained of severe headache; her eyes were suffused; her tongue coated, dry, and brown; her skin covered with a copious rubeoloid eruption; her pulse numbered 130, and respirations 26.

She was ordered ammonia, with chloric ether, and two drachms of brandy every hour.

At night, she became delirious; and the delirium lasted through the next day, becoming worse at night, so that she could with difficulty by kept in bed. Her head was shaved and a blister applied. The other symptoms continued much the same as on admission, the pulse and respirations rather increasing in frequency.

On the 11th, as she continued wild and delirious, two doses of morphia were given, which procured her some sleep. The following day there was less active delirium and more drowsiness. She was slightly purged, for which an opiate enema was administered; and as exhaustion seemed increasing, an enema of quinine and beef tea was ordered every two hours.

On the 13th, she was no better, but remained in the same drowsy state; her breathing was hurried, and there was some recurrence of diarrhœa. Opium was added to each quinine enema, and an ounce of brandy given every half hour: turpentine stupes were also applied frequently to her belly and chest. On the 14th, her pulse and respiration had risen to 140 and 44 and the next day she died.

On examining the body after death, the lungs were found [illegible] be much congested, and the lower lobe of the right lung solidified, exhibiting a carnified rather than a hepatized appearance.

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[1] From the notes of my clinical clerk, M.

The brain substance appeared healthy: there was a little fluid under the arachnoid.

The spleen was greatly congested, and broke down easily under pressure.

In the treatment of these local congestions, you will do well to keep in view their nature: that they are but symptomatic of the general disorder, the direct results of the vitiated state of the blood; and that with a return of the latter to its normal condition, a resolution of the congestion may be expected. I would not, therefore, advise you to be very anxious to adopt any specific measures beyond those which I have recommended for the treatment of fever cases generally: the due support of the patient by suitable food, and stimulants proportioned to the exigencies of the case.

The local treatment need not on this account be neglected. You will find as the most efficacious, and the least likely to be injurious, free counter-irritation by turpentine fomentations and occasional blisters of good size, and applied at various parts, such as we employed in the cases of Copstock, Church, Turner, and Selby.

The same cause which operates in the production of pulmonary congestion, is often effectual in producing a congested state of the brain, though of this almost the only evidence we have is derived from the post-mortem examination of fatal cases; for the only symptom of a congested brain occurring during life, with which I am acquainted, is a soporose condition bordering on coma; but this might very well and generally does result directly from a poisoned condition of the brain itself, and not from a mere increase or stagnation of blood in the organ. It must not be lost sight of, that much of the congestion of the brain observed after death, is due to the mode of dying. When the breathing is hard, when the moribund state is tedious, and above all, when the patient has been convulsed just before death, the greatest degree of congestion may be expected.

I will quote two cases illustrative of the greatest amount of morbid change which you are likely to find connected with the brain in those who have died of fever. In one we found the congested state marked by some darkening of the gray matter, and the occurrence of numerous bloody points in the white; while in the other a similar state had resulted in effusion into the ventricles.

CASE XXXI.[1] (Vol. xli, p. 6.) Charles Andrews, a painter, of intemperate habits, but generally good health, was taken ill on the 1st of May, 1853, with shivering, followed by heat and perspiration. The following day he had what his wife described as a fainting fit, and was insensible for some minutes. From the commencement of his illness, until his admission to the hospital on May 7th, he vomited constantly, and was also much purged.

When admitted, he complained of feeling drowsy and confused; considerable muscular tremors were present; he had almost entirely lost his memory, and showed a tendency to delirium. These symptoms, with the dry lips, furred tongue, suffused eyes, hot and dry skin, covered with an eruption of light-colored spots, resembling the eruption of measles, and the rapid pulse and respiration, told plainly enough the nature of his complaint, and the large dose of the poison which he must have received.

A mixture of chloric ether and ammonia, and half an ounce of brandy every hour, were prescribed.

The next day his pulse and respirations had fallen from 100, and 44, to 96, and 36, respectively; but the pulse was extremely feeble. There had been no recurrence of diarrhœa. At night, however, he passed five liquid evacuations; and an opiate enema was ordered. On the 10th, the brandy was doubled, his head shaved, and a mustard poultice applied to the scalp. His pulse rose to 116, and the respirations to 40.

During the 11th and 12th, the same symptoms continued and increased, the most prominent were restlessness and delirium at night, jerking of the limbs, general and excessive muscular tremors, and relaxed bowels, or ineffectual efforts to pass an evacuation.

The brandy was increased, and turpentine and catechu given, but he died on the morning of the 13th.

At the post-mortem examination "no effusion was found beneath the arachnoid, or in the ventricles; the membranes of the brain appeared perfectly healthy." "The brain itself was somewhat congested; the veins were turgid with dark blood; the gray matter of the convolutions was slightly deepened in color, and very numerous bloody points were seen on slicing it so as to display the centrum ovale." "The brain substance was hard and firm." The spleen was very soft, almost of a creamy or pulpy consistence.

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[1] Reported by Mr. Plowman.

In this case, the symptoms were due in my opinion to the *poisoning of the nervous matter* of the brain; in other words, to its perverted nutrition; and the fatal result was much hastened by the diarrhœa which showed itself so early.

Case XXXII.[1] (Vol. xviii, p. 9.) The other case is that of James Davis, æt. twenty-four, a man of temperate habits, who was admitted, May 13th, 1846, with fever. The commencement had not been sudden or marked by any shivering, but gradual, with languor and pains in his limbs, head, and loins. He had been ill for more than a week before admission, and during the latter part of the time, delirious.

On the day after admission, he was still delirious, passed some watery evacuations, complained of much abdominal pain, and was unable to empty his bladder, which became rapidly distended and had to be emptied with a catheter. Pulse 104.

His head was shaved; five grains of carbonate of ammonia were ordered three times a day, and half an ounce of wine every two hours, with beef tea.

The next day there was no return of purging, but the other symptoms continued and increased; his pupils were dilated, and he lay continually on his back. One ounce of brandy was now given, alternately with an ounce of wine, every hour.

On the 16th and 17th, he continued much the same; some rhonchus was heard in the chest, and his belly became tympanitic, and the mucous membrane of the mouth very foul with sordes. He died on the 18th.

In this patient, as in Andrews, there was much subsultus tendinum.

At the examination of the body after death, the membranes of the brain were found much congested, as also the white substance of the hemispheres, which was thickly studded with red points; the whole brain was softer than natural; there was a large effusion of pale straw-colored fluid into the ventricles.

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[1] Reported by Mr. R. D. Mills.

I have no doubt that in this case as in many others, the ventricular effusion was a passive dropsy consequent on the retarded cerebral circulation.

There were evidences of tuberculous disease of the lungs and mesenteric glands.

Peyer's patches were very prominent, and increased in size, but not ulcerated.

The subarachnoid effusions which we meet with now and then after fever are not of an active kind. They are the result of a certain shrinking of the brain, fluid being poured out to fill up space. Do not fall into the mistake of supposing that an effusion of this kind is instrumental in causing comatose symptoms. It, in truth, exercises no more than the normal pressure which seems a necessary condition of the brain's nutrition.

Of the cerebral symptoms—delirium, coma, and convulsions—two of which, at least, are of frequent occurrence in continued fever, I can only repeat what I said when speaking of the same symptoms in my lectures on rheumatic fever—that we have no grounds at all for supposing them due to any inflammatory or congested condition of the brain or its membranes, but must rather consider them as the result of that perverted nutrition which is the necessary consequence of the poisoned condition of the nutrient fluid.

With respect to the treatment of these symptoms, what I said in speaking of the treatment of pulmonary complications is applicable here also; you must treat them as part of the general disorder, not as distinct diseases. An increase of these symptoms, especially of the delirium, usually indicates an increasing exhaustion, and therefore demands a larger supply of stimulants.

Of local remedies, I find the application of blisters to the scalp and back of the neck, and the employment of a cold affusion either to the head, or over the whole body, the most efficient means of rousing a patient from a drowsy comatose state. The douche sometimes acts like a charm; it is most applicable to cases in which a lethargic state supervenes early, and before there is great exhaustion; and it should always be employed with as little distress to the patient as possible.

The three following cases are good examples of the occurrence of cerebral symptoms, and of the treatment I have recommended.

CASE XXXIII.[1] (Vol. xli, p. 197.) Zechariah Stilling, æt. 26, an Irish laborer, of irregular and intemperate habits, was brought to the hospital, February 11th, 1854, with unequivocal symptoms of fever,—a hot and dry skin, brown and furred tongue, some stupor, loose watery evacuations, numerous rose-colored spots, disappearing or becoming pale on pressure, and harsh respiration heard over the front of the chest. The attack commenced about nine days before with shivering, headache, and considerable diarrhœa.

Half an ounce of brandy was given every three hours, and beef tea; also some aromatic spirits of ammonia in decoction of logwood.

From the 11th to the 14th there was no change; his pulse continued 116, and the prominent symptoms were a noisy, restless delirium, with an obstinate determination to get up and leave the hospital, and great pain in his head.

On the 13th the brandy was increased to half an ounce every two hours; and on the 14th his skin was moist and his pulse had fallen to 100, and from that day to the 20th it gradually declined to 50.

On the 15th and 16th he was still restless and wandering, with pain and noise in his head. Mustard poultices were applied to the scalp. All the bad symptoms, however, rapidly passed off; the brandy was gradually reduced; and by the 28th he was fairly convalescent. There had been no return of diarrhœa throughout.

In this case the active stimulation had not been commenced sufficiently early. Had it been otherwise, the delirium would have been less developed.

CASE XXXIV.[2] (Vol. xxxix, p. 59.) John Selby, æt. 27, a detective police officer, of temperate habits and previous good health, was admitted to the hospital, December 11th, 1852, in a state of almost complete coma. It appeared, that about ten days previously he had been seized with severe rigors; he then grew rapidly ill, suffered from considerable purging, and, during the two days preceding his admission, was delirious. I will read you the report made by my clinical clerk of his condition on admission: "He appeared perfectly unconscious, and quite unable to swallow anything: when it was attempted to give him fluids to drink, they simply collected in the mouth, scarcely any passing into the œsophagus, and were slowly ejected by each expiration. His abdomen was covered by a great number of small, circular, somewhat purplish or mulberry-colored spots, having much the character of petechiæ, not raised, and not disappearing, though slightly fading at their circumferences, under pressure. There was no marked tenderness of the abdomen. Pulse 140, respirations 30."

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[1] Reported by Mr. Buzzard.

[2] Reported by Dr. Plowman.

Beef tea and half an ounce of brandy were administered every half hour, a mustard poultice was applied to the back of his neck, and a turpentine stupe between the shoulders. His head was shaved and a cold douche applied. The bladder was emptied by a catheter.

Great benefit seemed to result immediately from this free counter-irritation and stimulation; he was completely roused from his stupor and appeared altogether much better. On the following day, the 12th, the improvement continued; his pulse and respiration had fallen to 112 and 24 respectively; he had passed his water freely, though involuntarily, and his bowels had moved, but the motions were not relaxed. Some of the acetum cantharidis was rubbed on the scalp.

He passed a quiet night and slept well; but we were disappointed in the morning to find him decidedly weaker, his pulse risen to 120 and the respirations to 30, his tongue dry, with a thick brown coat. This was explained by the discovery that he had been neglected by the night nurse, who had omitted to give him the brandy and beef tea regularly. He passed a quantity of dark-colored acid urine, and some dark liquid fæces, voluntarily. The brandy was doubled, and a blister applied to the scalp.

On the 14th, after a comfortable night, he was again better, but his pulse remained the same, and he complained of some slight abdominal pain.

On the 15th, as there was some cough with a dark sanguineous expectoration, a turpentine stupe was ordered. By the evening his pulse and respiration had fallen considerably, numbering 100 and 22 respectively.

The following note was made on the morning of the 16th:—"He remains much the same. Has still a little cough, but the sputum is clear; breathing natural; the spots are becoming faded, and much fewer. Pulse 96; respirations 24. The tongue is tremulous but much cleaner; there is still no purging, and he does not complain of any particular pain in his stomach." The brandy was reduced again to an ounce every hour.

From this time there was steady improvement. The respiration and pulse declined; the latter was 92 on the morning of the 17th, and 88 in the evening; 84 on the morning of the 18th. His tongue became cleaner, his urine natural, and he passed good nights. An equal amount of wine was substituted for the brandy: that is to say, twenty-four ounces of wine were given him daily, and under this the pulse came down in the manner detailed.

There was something like a critical sweating on the 18th, which was also the 18th day of the disease: the perspiration was profuse, and continued through the 19th and 20th.

On the 21st he felt himself to be much stronger. His pulse was 80, full, and fairly strong, though still compressible; respirations 20. He only complained of some confusion of thought, and of a swimming sensation in his head, which disappeared in the course of a week or ten days, as his strength returned.

The amount of wine was gradually reduced, and on the 28th a quinine mixture was ordered, and on this treatment, with a liberal diet, he made an excellent recovery. He remained in the hospital until January the 22d, when he was discharged quite well and in almost his former strength.

CASE XXXV.[1] (Vol. xxxix, p. 213.) Thomas Keen, æt. 33, was admitted March 26th, 1853.

Ten days before he had been seized with shivering, and for a week afterwards suffered from relaxed bowels; but, from the time of his admission until he left the hospital, he was quite free from any recurrence of looseness or abdominal symptoms.

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[1] Reported by Dr. Plowman.

He was ordered some carbonate of ammonia with henbane in effervescing mixture, morphia at night, and an ounce of wine every two hours, with beef tea.

During the night of the 26th he was delirious, and had not recovered his consciousness by the morning of the 28th. His face was then flushed and his eyes suffused, his skin hot and dry, and the tongue presenting a brown central band. Pulse 92. On his back and abdomen were scattered a few distinct, non-elevated, rose-colored spots, rather smaller than a split pea, and some of them entirely disappearing on pressure. A great number of sudamina were also present on the belly at the upper part. He coughed frequently, and rhonchus and sibilus were heard in front.

On the 29th, there was more wandering, with drowsiness and slightly stertorous breathing. Pulse 120; respirations 28. The other symptoms were but little altered.

On the 30th, brandy was substituted for wine. He appeared to derive benefit from the change, for he slept better, and was less delirious and more conscious.

On April 1st, the brandy was doubled, *i. e.*, an ounce was given every hour, his head was shaved and a mustard poultice applied. On the 2d, a cold affusion was administered. On the 3d, there was a fall in the pulse from about 100 to 86, notwithstanding twenty-four ounces of brandy daily; and on the 4th, which was the 21st day of the fever, it did not exceed 72. He was then sweating profusely, the moisture running off his forehead.

On the 5th, the pulse was as low as 52. The brandy was reduced to four ounces, and porter and quinine given. On the 12th, he was convalescent.

In a former lecture, I spoke at length of the exhausting diarrhœa or hemorrhage which is apt to accompany cases of typhoid fever, and also of the morbid appearances found after death in the intestine. I shall have to allude to this intestinal hemorrhage again, as one of the critical discharges by which the fever sometimes terminates.

There is another abdominal symptom for which you must be prepared. The affection to which I allude is a form of tympanitis, or meteorism, as it is called. It consists in an inflation of the intestinal canal by gas more or less rapidly generated within it. In consequence of this inflation, the belly becomes very prominent, tense, and drummy, and is highly resonant on percussion at every point. There is no sense of fluctuation present, excepting when there may have been liquid diarrhœa, and a considerable quantity of fluid remains in the bowels. Under these circumstances an obscure fluctuation is perceptible, which you must not allow yourself to be misled into supposing to arise from fluid in the peritoneum. That the fluid is within the bowels along with the air, is proved by the borborygmi and other metallic sounds, which are audible under the influence of the peristaltic action of the bowels, or under strong pressure or succussion of the abdominal walls.

This distended condition of the bowel is due to a secretion of air from the mucous membrane, partly also, possibly, to a generation of gas from decomposition of the contents of the gut. There is no doubt a very defective nervous influence, which regulates imperfectly both the secretions and the muscular motion of the bowel.

This *meteorism* occurs, so far as my experience teaches me, in all the forms of continued fever, and does not, as one might suppose *à priori*, especially belong to that in which the bowels are so apt to be irritated, namely, the typhoid. On the contrary, I should say, it is of more frequent occurrence in typhus. I may remark that this symptom has not been nearly so often met with in my own practice, since I have adopted the plan of thoroughly upholding my patients from the commencement.

This tympanitic state is by no means peculiar to typhus or typhoid fever. It occurs in other diseases of defective nervous influence: in severe diseases of the spinal cord; in affections of the brain, such as acute meningitis, and in peritonitis. In all such cases, the influence of the intestinal nerves must be impaired; the muscular coat of the bowel must in great degree lose its tone, and allow the bowel to become full and distended,
the want of the resistance which a strong muscular coat
to the accumulation of gas.
which we adopt for this condition in typhoid

cases consists in the external application of turpentine, in the form of hot fomentations to the belly, the frequent use of enemata with confection of rue and turpentine, sometimes a mild warm aperient, and in extreme cases, galvanism.

Another incidental feature of fever cases, allied to the last, is a more or less perfect paralysis of the bladder, so that when it becomes full no active contraction of the detrusor muscle occurs, and the sphincter remaining closed, the urine is retained, and the bladder becomes distended. If this is allowed to continue, the urine will dribble away, and it may be long before the bladder recovers its muscular power.

We had illustrations of this condition in the patients Selby and Davis. Let me advise you when attending a case of fever to make frequent inquiry respecting the condition of the bladder, and if you can feel it forming a tumor above the pubis, to draw off the water with a catheter as soon as you can. I can say of this symptom, as of tympanitis, that under good support and stimulation from the commencement, it is of far less frequent occurrence.

An albuminous condition of the urine is occasionally found in cases of continued fever. When it occurs, we must consider it owing to a congested condition of the kidneys, arising not only from the general tendency to capillary congestion, but possibly also from a functional effort on their part to eliminate some of the poison from the system. The urine of the patient Church (Case XXV) was very albuminous, and revealed *blood casts* under the microscope. When you find albumen in the urine, you will naturally suggest to yourselves, is this due to diseased kidney or to a temporary congestion? The answer cannot be given at once: to discover any morbid change you had better wait till the fever has gone off. Meantime no harm is experienced by the passage of the albumen, and if there be evidence of morbid change, it must be dealt with when the patient has thrown off the fever.

I have yet a few remarks to make on the manner in which cases of fever terminate. You have all heard of the *turn* of a fever: the idea is an old one, as old as Hippocrates, that fevers
are wont to change suddenly for better or wor
called critical days. In later times t
little better than an old wife's fab

tions go far to establish the truth of it. The *crisis* of a fever is frequently marked by the occurrence of some copious evacuation, either of a natural kind, such as free sweating, which is by far the most common, or by some unnatural one, such as a profuse bronchial secretion of watery mucus, or the passage of a quantity of blood from the bowels. This is followed or accompanied by a rapid diminution of the febrile symptoms, and in favorable cases by speedy general amendment. This is not, however, invariably the case: a well-marked crisis may occur, and perhaps be followed by an abatement of fever, and yet from that time the patient, instead of recovering, may grow more comatose or exhausted, and the case terminate fatally.

I will give you three examples of well-marked crises, one of which was unfavorable.

CASE XXXVI. (Vol. xxvii, p. 164.) William Brown, a painter, twenty-nine years of age, who had been living badly for some time, towards the close of a week of unusually hard work, felt weary and indisposed, and on Saturday night was attacked with a fit of shivering, feeling alternately hot and cold; he continued chilly and shivering all Sunday, and was admitted into the hospital on the next day, Monday, May 28th, 1849, suffering severe pain in his head, back, and extremities, and with all the symptoms of fever—heat, thirst, loss of appetite, furred tongue. After having a warm bath there was some perspiration, and a copious eruption of spots, called in the record of the case, *petechial* (?), was observed. His pulse numbered 100.

A saline mixture of citrate of ammonia, and nitrate of potass was ordered, and a very small allowance of wine with plenty of beef tea.

The fever continued high for four or five days; he became deaf, stupid, light-headed; he coughed a good deal, and the eruption remained fully developed. The wine was increased to half an ounce every two hours.

On the 2d of June (the seventh day of the fever) his bowels became relaxed, and he passed a watery evacuation; this was followed, the next day, by a remarkable cessation of fever, the pulse falling to 60. He seemed, nevertheless, so extremely low, that eight ounces of brandy were ordered to be given in the next twenty-four hours; an enema was also given to check the bowels, and a large mustard poultice applied to the belly. From this time, however, the amendment was rapid, and he was discharged on the 14th of June, the twentieth day from the shivering.

In this case the purging seemed to mark the crisis.

CASE XXXVII. (Vol. xl, p. 47.) Jane Green, thirty-eight years of age, from the parish of St. Giles, was admitted to the hospital, May 24th, 1853, with fever. She stated that she had never had a serious illness, but that her habit had been to live badly, and drink hard.

Her illness began, May 14th, with shiverings, perspirations, numbness of the hands and feet, deafness, relaxed bowels and nausea; with a hard cough and sense of weight in her chest.

She first applied at the hospital as an out-patient, but becoming much worse she was taken in.

The following was her condition on admission: "Her pulse was 140, her skin very hot, her arms and chest dotted all over with red spots, and her tongue and teeth were covered with some blackish blood which she had vomited just before. Her bowels had been recently moved, and the motion was dark-colored and offensive."

She was ordered five grains of carbonate of ammonia and fifteen minims of chloric ether every three hours, an ounce of brandy every hour, and a morphine draught at night. Her head was shaved, and a mustard plaster applied to the chest.

On the next day, the 25th, an enema of ten grains of quina in two ounces of beef tea was ordered every two hours, and the morphine draught to be repeated at night.

On the 26th, she appeared much better, complaining only of thirst. She slept a good deal, but wandered at times; pulse 110; her urine was found to be albuminous, and the skin on the buttock seemed threatening to slough.

Acetum cantharidis was applied to the scalp, so as to produce vesication, and the enema of quina and beef tea continued every four hours.

In the evening, *she was purged three times, passing very dark and offensive motions*, for which a starch and opium enema was prescribed.

On the 27th, she seemed better; her pulse was 108, her tongue red and moist, the quinine enema was discontinued.

On the 28th, the fifteenth day of the fever, the pulse had fallen to 90, and the patient was sweating profusely.

The brandy was reduced to half an ounce every hour.

On the 30th, the pulse was 84; and on the 3d of June 75. Her tongue was then clean, and a chop was ordered and wine instead of brandy.

In this case, although great care had been used, a sore had formed in the gluteal region, which required poultices and afterwards stimulating dressings, and detained her some weeks in the hospital.

Case XXXVIII. (Vol. xliv, p. 81.) Sophia Bruce was admitted with fever July 26th, 1854. There had been two or three cases of fever in the house from which she came. Her illness had commenced with shivering, and the usual symptoms, seventeen days before her admission. Shortness of breath had occurred very early, and formed a prominent symptom when she was admitted; crepitation and rhonchus were then also heard both in front and behind; the usual febrile symptoms were present, and an eruption of scattered rose spots on the abdomen.

Half an ounce of brandy was given every hour, also chloric ether and ammonia, and enemas of salicin and beef tea; and turpentine stupes were applied to the chest.

She continued much the same for some days. Her breathing was extremly rapid, exceeding 50; her pulse about 100; but the heart's action on the 29th was nearly twice as rapid as the pulse. The brandy was doubled, and quinine substituted for salicin.

On the 30th, the 21st day from the shivering, *she was sweating very profusely;* but from that day she become worse, and on the 31st, was semi-comatose; some purging also occurred. These symptoms continued, the diarrhœa with some intermissions, until her death on the 9th.

After the sweating on the 30th, the fever seems to have abated; for on the 3d of August, we have the following note, "Her skin is cool and moist; the fever seems to have abated." It appears to me that the cause of the fatal termination was the excess of the critical discharges, which with the diarrhœa exhausted the patient. No doubt the attempt at relief by the natural process may occur, unsuccessfully. And herein we learn the importance of upholding patients from the commencement, that these critical evacuations may not produce fatal exhaustion. Sometimes a crisis, though favorable, is marked by a temporary exacerbation of the symptoms, as in the following case:—

Case XXXIX. (Vol. xli, p. 213.) Daniel Shea, æt. twenty-six, a laborer, of intemperate habits, was attacked with violent pains in his head and limbs. He continued to have headache for a week, and passed disturbed and restless nights, until his admission to the hospital, March 15th, 1854. He was then suffering from fever; his pulse was 106, and respirations 30; his tongue furred; his whole body, but especially the back and belly, was covered with a papular rose-colored eruption, the spots being aggregated into patches of variable magnitude, and disappearing on pressure. Half an ounce of brandy was given with beef tea every two hours, also ammonia and chloric ether.

For about a week, he continued very ill, with little change in the symptoms; his pulse continued feeble and rapid, 112–104; his tongue dry and furred, and lips black with sordes. For several nights he was delirious; the brandy was doubled, and ten minims of tincture of opium were added to each dose of his mixture. Subsultus tendinum, cough, and relaxed bowels occurred, but did not continue.

About the 21st and 22d, delirium gave place to deafness and drowsiness, the eruption disappeared, and his pulse fell to 100. At night, however, he became very delirious, and on the morning of the 23d, the 20th day of the fever, he was unconscious: he lay on his back with his eyes nearly closed, and his face covered with a profuse sweat. His head was shaved and blistered.

This condition of the patient might naturally enough have led to an unfavorable prognosis; but from this time he began to amend, the pulse steadily falling, so that by the end of a month from the commencement of the attack he was quite convalescent.

A crisis most frequently occurs in this country, I think, speaking roughly, at the end either of the 2d or 3d week from the shivering, more commonly the latter, not often much earli later, but sometimes on intermediate days.

The case of Copstock (XXIV, p. 138) affords a good illustration of crisis by free sweating.

Dr. L. Traube, of Berlin, has published the results of some elaborate researches, made by himself, in an essay on crises and critical days, of which you will find a review by Dr. E. H. Weber, in the *Medico-Chirurg. Review*, January, 1853. The investigations were made with special reference to the temperature of the body in febrile affections, that being taken as the index of fever. He finds that crises, marked by a fall in temperature and such critical evacuations as I have mentioned above, occur, in continued fever, most frequently on the 5th, 7th, 9th, or 11th days; that is, on the odd days only, and considerably earlier than usually happens in this country. The days are successive intervals of twenty-four hours, commencing from the first accession. But the difficulty of determining the exact period of accession is so great, that the assignment of the crises to the odd days with accuracy must, in many cases, be quite impossible.

Very often these febrile symptoms subside gradually without the observation of any critical evacuation. This is what has been designated resolution of the fever by *Lysis*. The occurrence of such cases in no degree invalidates the notion that the fever may be quickly terminated by a critical discharge, for it is quite possible that that, which under other circumstances would be limited to the skin or the kidneys or the bowels, in such cases is distributed among these emunctories, so that the increased discharge from each becomes comparatively insignificant, and escapes detection.

I shall conclude this too long lecture by a few instructions to you, which for conciseness I have put into an aphorismic form, touching the management of the kind of cases upon which I have been commenting. Let me, however, first make this remark, that however important it may be for clinical purposes, and for prognosis, and for satisfying the inquiries of friends and relatives, to make the distinction between typhus and typhoid, there is no essential difference in the treatment, excepting that in the latter you must be always on the lookout for bowel disturbance, and take measures to prevent or to check it.

My first rule is this,—never give up a fever patient until he is plainly *in articulo mortis.* Patience, perseverance, and steady adherence to a well-devised plan of treatment are, in fever cases, often crowned with success under the most discouraging circumstances (case of Shea, No. XXXIX, vol. xli, p. 213).

2. When you undertake a case of this kind, insist upon having a good nurse; give her full instructions, if possible in writing, and require her to keep a note of all food and medicine administered by her. Do not trust to the nursing by relatives.

3. It is not advisable that you should see your patient too often; once, or at most twice a day, will be, in general, quite sufficient.

4. Do not be anxious to account or provide for every new symptom or change that may arise. Do not treat symptoms too much, but look to the general condition of your patient. Diarrhœa, delirium, coma, hemorrhage, are the symptoms which you should look out for and be prepared instantly to meet.

5. Watch the pulse closely both as to quality and frequency. Always keep in view that increase of quickness is a sure sign of increasing debility, and that diminution of quickness (when not referable to any cerebral affection) is the reverse.

6. Never allow a day to pass without carefully examining the abdomen of your patient, and especially the region of the bladder.

7. Do not be anxious about the action of the bowels, and the so-called secretions. Many a patient in fever has fallen a victim to the *diligentia medici nimia,* in improving "the secretions." You may make them perfect in color and consistence, and yet your patient will die.

8. Restrain diarrhœa and hemorrhage, and when, in typhoid fever, you have fairly locked up the bowels, keep them so. Patients will go for four or six days, or even longer, without suffering inconvenience from this state of constipation.

9. From the first moment of your attendance let it be your constant and anxious effort to uphold the vital power of your patient, by nitrogenous food given as broths, and carbonaceous food, selected from farinaceous substances, and from alcoholic fluids, such as wine, brandy, or other fermented liquors.

10. Increasing delirium and coma are signs of increasing debility: both indicate the necessity for additional support; coma is often benefited by freely blistering the nucha and scalp, and even the region of the heart.

## LECTURE VI.

### *On Erysipelas.*

I TAKE as the subject of my lecture to-day, gentlemen, two cases of erysipelas which have lately been under treatment in the hospital. This disease deserves your most diligent watching, inasmuch as it is of frequent occurrence in this town, and one which it is very important you should be well prepared to treat. One of the cases to which I shall refer is a good example of that form of erysipelas which is most commonly met with in the medical wards,—erysipelas of the head and face; the other, of that form which comes more immediately under the notice of the surgeon, though in both instances the disease is essentially one and the same.

CASE XL. (Vol. xliv, p. 189.) The first case is that of John Child, who was in No. 4 ward, but who has now left the hospital; this patient presented a good example of an average case of erysipelas of the head and face, coming on, as it very often does, after exposure to wet and cold. His history is as follows:—He is a laborer, twenty years of age, and has always enjoyed good health until a few days before his admission into the hospital, when, after considerable exposure to wet and cold, he was seized with general febrile symptoms of a severe character, and these were accompanied with swelling and redness of the nose, which gradually spread, until they involved the whole of the face and head.

Unfortunately, our notes of the case at this period are not so full as could be wished, and we are, therefore, quite in the dark with respect to certain points in the early history of the attack, with which it is important that we should be acquainted. My clinical clerk, for example, has omitted to mention whether miting or rigors were present among the first symptoms.

Erysipelas generally begins with rigors, and the attack is also often ushered in by vomiting; indeed, vomiting is a common symptom at the onset of the disease; and to such an extent does this obtain, that whenever I meet with a patient who has been suddenly taken with vomiting, and this vomiting accompanied with, or preceded by, rigors, I deem it expedient to watch carefully for erysipelas.

Sore throat, too, is often one of the earliest symptoms of this malady; the disease sometimes appears to begin at the fauces, which under these circumstances are generally much redder than natural; and from this part it seems to spread outwards through the nose, affecting the alæ nasi, the face, the eyelids, the forehead, and lastly, the scalp,—following, in fact, a regular course; and this was very much what occurred in the case under consideration. At other times the throat and face become affected simultaneously.

The swelling of the face is frequently so great that the eyes are, to use a vulgar expression, "bunged up," and the patient is quite unable to open them; and so they generally remain for some days, a copious secretion, poured out by the conjunctiva or Meibomian glands, concreting, and gluing the eyelids together. In this instance, the swelling rapidly spread, the face became greatly puffed up, and the eyelids completely closed; and in this condition the patient was brought into the hospital. Under the treatment adopted, and which I am anxious to recommend to your particular attention, the fever diminished in the course of four or five days, the swelling of the head and face subsided, the patient was able to open his eyes, and the pulse fell in frequency, and in six days after his admission he was fairly convalescent, "sitting up, and able to move about the ward."

The duration of the fever in erysipelas is not long, usually varying from seven to fourteen days, but the phenomena of the disease naturally divide themselves into two classes,—the *primary* and the *secondary*. The former of these comprises the rigors, the vomiting, the general febrile symptoms, the rapid pulse, the peculiar redness and swelling of the skin, &c.; and if due attention be given to uphold the powers of the patient from the commencement of the attack, it rarely happens that the secondary phenomena manifest themselves at all. The poison of erysipelas, whatever its nature, appears to fall chiefly upon the tegumentary system, expending its virulence upon the true skin and the mucous membranes; yet it frequently happens, when the march of the disease has not been successfully opposed by well-directed measures, that it spreads from the true skin and mucous membranes to the areolar tissue beneath these structures, and it then gives rise to the secondary phenomena of the malady, namely, fever of the hectic kind, and the formation of collections of pus, either at the seat of the primary disease, or in other parts of the body. It is very important that you should keep in view this tendency of erysipelas to involve the cellular tissue beneath the skin, and there induce the formation of pus; for it should be your aim to cure the disease without allowing it to bring about these secondary consequences, which are always exceedingly wearying and debilitating to the constitution, and sometimes so exhausting as to terminate the patient's life. In the case which I have just related to you, the cure was speedily effected without the occurrence of any of these secondary results; and this I attribute, in great measure, to the plan of treatment which was adopted.

Case XLI.[1] (Vol. xlviii, p. 22.) In the second example of erysipelas which I wish to bring under your notice to-day, the result was by no means so satisfactory; indeed, the case forms a good contrast to that of John Child, which you have just heard, illustrating, as it does, the less favorable course of the disease, under a very different plan of early treatment from that pursued in his case, as you will presently see.

The patient is a lad named John Walker, who was admitted into Fisk ward, February 17th, 1855, with erysipelas of the right leg and foot; and, I am sorry to add, he is still under treatment in the hospital, and likely to be so for some time to come. There was no history in this case of the patient's having received any injury, and nothing whatever to explain the occurrence of the attack; but it was attributed by the child's parents to his having lived very badly for some time, and to his having been of late much exposed to wet and cold. The short account which we have of the case is this: About a fortnight before the lad's admission into the hospital, it was noticed that his right leg, all the way down to the foot, was exceedingly red and some-

[1] The record of this case was kept by my clinical clerk, Mr. Jenkins.

what swollen, and this was accompanied with general febrile symptoms; but neither his parents nor himself could remember whether he had any shivering or not. He was kept in bed, and the only treatment which was adopted consisted in the application of hot fomentations to the limb, and from the necessitous circumstances of the parents, in the administration of the poorest kind of food, and very little of that.

Upon his admission to the hospital on February 17th, about fourteen days after the commencement of the attack, it was found that the primary phenomena of the disease had run their course, and that the secondary phenomena had supervened. For this we cannot attach blame to the medical treatment adopted, for, properly speaking, he had none; but it is rather to be ascribed to the extremely bad living and poor food upon which the patient had subsisted during the whole of the winter, and to his having been deprived, during the first fortnight of his illness, of those comforts which are so necessary during a severe attack of this disease. And here let me call your attention to the fact, that if low diet, and that general course of treatment which is commonly summed up in the word antiphlogistic, be essential to the cure of erysipelas, surely this poor lad had enough of it! Nevertheless, it did not succeed in preventing the secondary phenomena of the disease from manifesting themselves; for, upon the patient's admission, there was distinct evidence of suppuration beneath the integument of the leg, for the relief of which it was necessary, after a day or two, to make two incisions through the skin, one along the outer, and the other along the inner side of the ankle; and the following day it was thought expedient to make a third incision along the dorsum of the foot, for the escape of a considerable quantity of pus which had collected in that situation. The weakness of the patient on his admission was extreme, and his pulse was 120, small and very compressible; the treatment upon which he was at once put, consisted in the free exhibition of beef tea and brandy, ammonia and chloric ether.

The further history of the case is as follows: The erysipelatous swelling of the leg and foot subsided, but a very free discharge of pus continued, and still continues, to take place; the rapidity of the pulse kept up, and the exhaustion became, if possible, still greater than on the patient's admission, so that it was found necessary to adopt every means which could be resorted to for the purpose of upholding his strength. To this end, beef tea enemata, each containing ten grains of quinine, were administered, regularly, every fourth hour; half an ounce of brandy every half hour; and as much beef tea, and milk thickened with flour, as the patient could take. Notwithstanding all this, the pulse kept up, being 134, 137, 140, &c., and never below 120; the suppuration continued to progress, and, a day or two ago, a large sinus was found to have formed between some of the muscles of the leg, so that it is even now exceedingly doubtful whether this lad will ever recover; for, when suppuration has taken place to this extent, it often not merely spreads along the areolar tissue between layers of muscles, but also, when in the neighborhood of joints, it may so extend as to involve these also. It is by no means improbable that some of the joints of the tarsus may here become affected, and render the amputation of the leg necessary; and such a measure as this, I need not say, would be attended with great hazard to life in a patient so thoroughly depressed and exhausted as this poor boy is.

Before dismissing the subject, let me observe, that in all these cases, the rapidity of the pulse from day to day is a valuable index of the progress which the suppurative process is making; for if you find that the rate of the pulse keeps up, despite of free supplies of nourishment, you may infer that more mischief is about to ensue, and that further suppuration will occur; and under these circumstances, it will seldom happen that the pulse will come down, until a free discharge of purulent matter shall have taken place.

Such then, gentlemen, are the two cases which will serve as the basis of my remarks to-day. The one, an ordinary case of erysipelas of the head and face; the other, a case of that form of the disease which is most frequently met with in the surgical wards,—phlegmonoid erysipelas, and which is precisely analogous to traumatic erysipelas, or to that which follows a surgical operation.

Now, as I have already remarked, the most common form of erysipelas which comes under the care of the physician is that of the head and face. Let us trace the clinical history of this disease. It very frequently begins in the throat, and it is important that you should be acquainted with the various courses which it may take. It is one of the features of this disease that it has a remarkable tendency to spread, sometimes wandering all over the body, from face to neck, and neck to trunk, being then termed *erratic* erysipelas. When, then, it has commenced in the throat, it seldom finishes its course there, but immediately begins to spread, and its ordinary course is upwards from the throat, through the nose, to the face and head; sometimes it pursues the opposite direction, and wanders down, over the respiratory mucous membrane. In following this latter course, it generally passes very quickly (most fortunately for the patient) over the laryngeal mucous membrane, and then, affecting the mucous lining of the air-passages below the larynx, produces *erysipelatous bronchitis*—a complaint of more common occurrence than is generally supposed, of very fatal tendency, accompanied with great prostration, and which often leads rapidly to purulent expectoration. When this malady terminates fatally, it generally does so by inducing that condition, which, since the death of the Emperor Nicholas of Russia, has been so much talked about as paralysis of the lungs—a bad term, intended to express that state of things which occurs when the air-passages become choked with mucopurulent secretions which the patient is too much exhausted to expectorate. This accumulation of viscid secretion in the air-tubes tends, of course, to produce suffocation, by interfering with the proper aeration of the blood in the lungs; and the consequent retention of carbonic acid in the circulating fluid poisons the nerves and nervous centres, diminishing their excitability, and rendering less free those reflex actions on which expectoration in some degree depends. Thus the accumulation of the secretion in the air-passages is still further promoted, and so these two conditions, narcotized nerve and loaded air-tubes, go on mutually acting and reacting upon each other, until at length death takes place.

I shall illustrate these different courses which erysipelas takes by two or three examples from my hospital case-book. I have said that erysipelas of the head and face frequently begins in the throat; the following case commenced in that way, but the throat affection was more than usually severe, and attended with redness and difficulty of swallowing.

Case XLII.[1] (Vol. xxx, p. 126.) John Lawrence, fifty-one years of age, of rather intemperate habits, after suffering from a bad sore throat for more than a week, with great restlessness, was attacked with vomiting, shivering, and headache, and these were followed, the next day, by erysipelas of the face.

He was admitted on the 2d of April, 1850, the fourth day from the shivering. His nose was then of a deep crimson red, and this extended to the eyelids; there was much œdematous swelling and great pain, and he suffered from faintness and sickness. His pulse was 86. A dose of the hospital magnesian aperient mixture was ordered, and liquor ammoniæ acetatis with excess of carbonate of ammonia.

On the 3d, the day after admission, the erysipelas had not extended on the face, but an erysipelatous blush was observed over the fauces; he had been unable to sleep in consequence of the pain in his head, which continued very severe.

Ten grains of the compound ipecacuan powder were ordered.

On the 4th, after a sleepless night, his throat was extremely painful, and he was unable even to swallow his saliva. Chloric ether was now given, and between two and three drachms of wine every hour. After this the erysipelas subsided in his face, but he still complained of acute pain in his head, and this, and the throat affection, remained the most prominent features of the case. His pulse throughout had not exceeded 88. A stimulating gargle was ordered for his throat.

On the 9th, he commenced taking two grains of quinine every four hours, and on the 12th some morphia at night. At the end of the first fortnight a very great improvement had taken place, and he left the hospital, about three weeks after his admission, quite well.

The next case is an example of erysipelas of a very erratic type, beginning in the trunk and wandering at length to the neck and head.

Case XLIII.[2] (Vol. xvii, p. 100.) Maria Marshall, a married woman, aged thirty-seven, was admitted into the hospital in a state of general ill health: she was pale, weak, and low, with some enlargement of liver and œdema of the lower extremities.

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[1] Reported by Mr. Dickinson.

[2] Notes of this case were kept by my clinical clerk, Mr. (now Dr.) Tanner.

She was doing well until leeches were repeatedly applied over the liver. After the last application, July 17th, 1846, she was attacked with shivering; she had several rigors during the day, and towards evening became delirious. On the following morning an erysipelatous redness was observed extending from the leech-bites up to the right mamma and shoulder: she had a hot, dry skin, much thirst, a thickly furred tongue, a high pulse (100), and an anxious face.

The erysipelas continued to extend. On the fourth day some carbonate of ammonia was ordered, also a little wine, and repeated hot fomentations. The next day, the wine was increased to half an ounce every two hours.

The inflammation now extended to the patient's back, and assumed an erratic character. From the back it spread to the neck, and thence to the face, accompanied with great redness and swelling. There was some delirium and continued fever.

By the 3d of August, about the eighteenth day, she was very much better; free from fever and pain, and the erysipelas was rapidly subsiding. This improvement continued; but she was left very deaf and weak. She was, however, greatly benefited by a tonic regimen, and left for a convalescent institution on the 31st.

A third case I shall bring before you was one of traumatic erysipelas, in which severe dyspnœa and bronchitis suddenly supervened, and terminated the patient's life in a few hours.

CASE XLIV.[1] (Vol. xxxiii, p. 27.) Edward Gresley, æt. 50, was admitted into King's College Hospital, under Mr. Fergusson's care, for hæmatocele. An operation was performed on the 26th of December, 1851; and the patient continued to do very well until the 8th of January, when he became very low, delirious, and generally ill. Opium and brandy were given very freely. Erysipelas soon became apparent about the wound, and rapidly spread over the groins and thighs, and some way up the back.

On the 17th, the redness was less, and he seemed generally relieved; on the 20th, it had nearly disappeared. He continued doing well until the night of the 26th, when he was attacked with severe dyspnœa, and intense catarrhal sounds were heard in the bronchial tubes on listening to the chest. For this condition he was placed in Rose ward, under my care, on the 27th; his pulse was then 148, and the respirations 28; intense rhonchus and crepitus were still heard throughout the chest.

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[1] Reported by Mr. (now Dr.) E. Liddon, Physician to the Taunton Infirmary.

The treatment consisted in giving two drachms of brandy every half hour, with strong beef tea, also chloric ether and ammonia every hour; and in the application of blisters to the front, and turpentine stupes to the back of the chest.

On the morning of the 28th, the same symptoms of severe bronchitis continued: his pulse and respirations were 128 and 32; the breathing had become even more difficult since the night, and the prostration was extreme. The turpentine stupes were continued, and the brandy increased to three drachms every half hour, and hot-water bottles were applied to the feet and sides.

Our patient soon became insensible and shortly after breathed his last.

On making a post-mortem examination, we found great congestion and œdema of both lungs, their surfaces being impressed by the ribs. There were some pleuritic adhesions. The bronchi were choked up with mucus, and the whole lung tissue was very friable, breaking down with but little pressure: this we attributed to a post-mortem change.

Incipient atheromatous disease was observed in the heart and great vessels.

This intense capillary bronchitis is a most intractable disease. I know of no treatment which affects it in the slightest degree. If you reduce your patient by giving tartar emetic, or by bleeding, or both, you intensify the œdematous condition of the lungs and accelerate the fatal termination. If, on the other hand, you administer stimulants freely, and nutritious broths, you may prolong life for a very brief period, but this is all. This capillary bronchitis occurs independently of erysipelas, and is often found in connection with an attack of influenza, the poison of which is probably similar to, though not identical with, that of erysipelas. It occurs sometimes in gout, and likewise in connection with the rapid development of tubercles in the lungs.

In some instances, the erysipelas commencing in the fauces spreads no further than the laryngeal mucous membrane, and it then produces what is termed *œdema glottidis*—one of the most formidable affections to which the human frame is liable. I cannot too strongly insist on the importance of keeping in mind the true pathology of this most alarming malady; for if you thoroughly understand the true nature of this complaint, and act up to that knowledge, you may, I believe, save many lives; whereas, if you are shilly-shally in your treatment, either from ignorance of the pathology of the disease, or from prejudice in not acting up to your knowledge of it, you will certainly lose your patient.

The disease consists in an erysipelatous inflammation of the laryngeal mucous membrane which rapidly leads to swelling of the glottis, and thus to such a degree of narrowing and occlusion of the rima glottidis, as induces the most intense dyspnœa, which is quickly destructive to life. For this condition there is but one way of affording relief, and that is by immediately making an artificial opening into the trachea, below the chink of the glottis; the further below this point the opening can be conveniently made, the better, though, as you are all doubtless aware, the lower down the aperture into the trachea, the more formidable is the operation. I would especially impress upon you that you should lose no time in resorting to this step, after having become satisfied as to the nature of the attack: the sooner the operation is performed, the greater probability will there be of your saving the patient's life. Nevertheless, I would remark, that although the first step in the treatment, the providing an artificial inlet for air into the lungs, which has to be performed by the surgeon, is a very important one, still the most important part of the treatment belongs to the physician; and any one who has witnessed the proceeding must have felt convinced, that although the relief afforded by the operation is instantaneous as regards the breathing, yet the patient will still die, unless proper means are adopted to combat the local affection, and the constitutional state which accompanies it.

I had long been convinced that the ordinary so-called antiphlogistic means are not only powerless as regards the œdematous state, but tend to depress the vital powers of the patient, already much lowered by the necessary surgical interference;

and I determined, when opportunity offered, to pursue, from the moment of the operation a different line of treatment.

CASE XLV. (Vol. xlii, p. 141.) There are many present who will remember to have witnessed a case of this disease which was in Rose ward last summer. The patient, Henry Wallis, a very fine, strongly built young man, twenty-two years of age, was brought to the hospital with marked symptoms of acute œdema of the glottis. The urgency of inspiration soon became extreme. Mr. Edwards, the house surgeon, with great promptitude and tact, performed the operation of tracheotomy. I determined, with the assistance of my clinical clerks, Mr. Teale and Mr. Holberton, and of many of the students, to whom I am greatly indebted, that nothing should be left undone to uphold the strength of the patient, and to keep a close watch upon him, through the kind co-operation of relays of men who sat by his bedside by two at a time; and I am happy to say, I have a most accurate detail of the case from the date of the operation to that of complete convalescence. He was fed upon strong beef tea, and brandy, of which half an ounce was given every hour; ammonia and chloric ether were also freely exhibited; and as at first his power of swallowing was not good, owing to the œdematous state involving the epiglottis, injections of beef tea, containing a considerable quantity of quinine, were thrown, at stated intervals, into the rectum. In short, this patient was well supplied with food and stimulants, but in small doses at short intervals; and, you will hardly believe it when I tell you, that from the beginning of this treatment he had scarcely any fever; his pulse from 110 in a minute on the day of the operation steadily came down to 99 on the first, 86 on the second, and 75 on the third day. On the evening of the second day, the tube was taken out of the trachea, as he was found to cough up mucus freely through the glottis; in a fortnight the wound was healing up kindly; and in a month from the date of the operation, the patient was discharged quite well, the wound being by this time perfectly healed.

Let me relate to you another highly interesting case, illustrative not only of the efficacy of this mode of treatment, but also of the evil effects of not continuing it steadily, and showing in a very marked manner how depressing to vital power is the influence of the disease itself.

CASE XLVI. A gentleman, sixty years of age, of spare habit, who had come to town for a short time, had a shivering, followed by hoarseness, amounting nearly to complete loss of voice, with difficult stridulous inspiration; soon followed by great difficulty of swallowing—each attempt at deglutition giving rise to a severe spasmodic cough. I saw him for the first time late at night. The voice affection had commenced, and the breathing was slightly stridulous at that time. On visiting him the next morning between eight and nine o'clock, I found the breathing and deglutition much more difficult, and the voice worse. I now felt it necessary to ask for surgical assistance, and Mr. Bowman was requested to see him. We agreed to pursue the treatment already commenced—namely, quinine injections every fourth hour, brandy half an ounce every hour, and such nourishing food as he could be got to swallow. We thought the operation might prudently be postponed until two o'clock in the afternoon, when it was arranged that we should meet again and avail ourselves of the invaluable judgment and experience of Sir Benjamin Brodie. At this meeting we found the patient still breathing with much difficulty, but deglutition was easier, the pulse was falling, and fever was less. Further postponement of operation was agreed to, and a third meeting appointed for seven o'clock in the evening. At this time, finding the symptoms still improving, pulse less frequent, deglutition better and breathing easier, we began to hope that surgical means might not be necessary, and this hope was confirmed by the still improved aspect of the symptoms at a fourth visit late at night.

On the following morning (the third day of the disease) all the symptoms were still better—deglutition had become comparatively easy, and the breathing sufficiently free to remove all anxiety as regards the risk of suffocation. The treatment had been steadily pursued through the night notwithstanding much opposition from the patient. It was continued throughout that day, omitting the enemata, and at the evening visit the symptoms were still improving, but the patient was much opposed to the frequent administration of food, and the consequent interruption of sleep, which I deemed necessary. Nevertheless, I left positive instructions that he should not be allowed to sleep more than two hours without food, until my morning visit. Immediately after I had left the house, he called the nurse to his bedside, and directed her in the most peremptory terms not to come near his bed during the night, saying that he would not submit to "doctors'" orders, and was determined to have a good long sleep. Unhappily the nurse obeyed; exhausted nature was not restored by sleep: on the contrary, he sank rapidly in the course of the night, and next morning I found him hopelessly prostrate. Nothing now seemed of any avail to arouse the failing powers of life, and in the course of the afternoon he became comatose, and died late at night.

You will, I am sure, allow me to add another case illustrative of the good effects of this treatment in œdema glottidis.

CASE XLVII.[1] (Vol. xlviii, p. 126.) Hugh le Fevre, æt. 36, was admitted with symptoms of œdema glottidis, July 24th, 1855. His occupation, that of a showman, had obliged him to make undue use of his voice, and he had repeatedly suffered from sore throat. About two years before, an unusually severe attack had occurred: his throat was then red and swollen, and he suffered from difficulty of breathing and pain in swallowing. The more acute symptoms passed off, but a chronic affection remained, and he continued subject to relapses. The attack which brought him to the hospital had commenced about a week before. No history of syphilis or other illness could be made out.

When admitted, he was breathing with great difficulty, thirty-two times a minute; his face was dusky and swollen, and he was evidently nearly suffocated.

Tracheotomy was performed by my advice: the breathing was instantly relieved, and the patient restored to comparative ease. After the operation the pulse and respirations were 80 and 28. Small quantities of brandy (ʒj) and beef tea were administered every quarter of an hour, and the tube was frequently cleared of mucus.

In the evening of that day his pulse rose to 90, but the breathing remained 28. He passed a comfortable night, and was only disturbed by occasional fits of coughing. Towards the next morning (July 25th) he had some sleep, and the pulse and breathing afterwards were 84 and 26. The brandy was reduced to half an ounce every two hours. In the evening the pulse and respirations were 86 and 24 respectively.

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[1] From Mr. Wharton Hood's notes.

During the two following days, the 26th and 27th (third and fourth days), he continued remarkably well, disturbed only at times by cough or sickness. On the 26th the pulse was 90, and the respirations 24; on the 27th they were 90 and 22.

By the 28th no unfavorable symptom had occurred; his pulse and breathing had fallen to 81 and 20, and the brandy was further reduced to half an ounce every four hours. Some redness surrounded the wound. The epiglottis, which had been swollen and stiff, felt softer.

On the 29th he was still doing well: the pulse and breathing were 85 and 24. On the 30th the pulse was 80, the respirations 29; on the 31st they were 70 and 24. He was now more troubled by cough, and there was increased mucous secretion. On the 30th five grains of iodide of potassium were ordered three times a day.

By the 3d of August (tenth day from the operation) he felt quite well: his cough was much better and his appetite good. On the 8th he sat up for three hours, and could eat perfectly.

On the 13th the tube was removed. His breathing became difficult in the afternoon, and in the evening the tube was returned, although with some trouble. The iodide of potassium was discontinued on the 16th. On the 27th the tube was permanently removed, and no further difficulty in breathing experienced. The brandy was discontinued.

He remained in the hospital until the 8th of September; by which time the aperture in the trachea had closed, he was quite well, and had recovered his voice.

Now in looking at the statistics of these cases, you will find that the deaths are but little under a hundred per cent.; indeed it is a rare thing for one to hear of a case of recovery from œdema glottidis after the operation of tracheotomy has been performed; and it seems to me very questionable, whether similar treatment to that which was pursued in these instances, ought not to be adopted in all cases of tracheotomy, for the operation is in itself a very severe one, and accompanied with great shock to the nervous system, while there is invariably great distress of the respiratory organs during its performance.

I gave you just now an instance of erysipelas of the face accompanied throughout by redness and soreness of the throat with difficulty of swallowing. There is yet another form of erysipelas which commencing in the throat confines itself entirely to the faucial region, inducing paralysis of the pharyngeal muscles with utter inability to swallow and great prostration; these cases are rare and of a very fatal tendency; far more formidable indeed than those in which the throat is only affected transiently, or in common with the skin of the face and head. I need not now enter into the particular consideration of these cases, as I intend to devote a lecture exclusively to the subject.

Thus, then, you see that erysipelas not only affects the external integument of the body, but also the mucous membranes, both alimentary and respiratory. And in addition to those parts which I have already mentioned as so peculiarly liable to be fixed upon by the poison of this disease, there is one other tissue which, I suspect, it not unfrequently invades, namely, the peritoneum, producing what is known as puerperal fever, or puerperal peritonitis; a disease which I believe is really of an erysipelatous nature, and which certainly in most, if not in all instances, requires to be treated upon a thoroughly supporting plan.

When erysipelas terminates the life of a patient, it most frequently does so by inducing a state of extreme prostration, and the patient sinks exhausted. This was what occurred in the man J. H. Collins (Case XLIX, p. 142), whose history I will presently read you. A second mode of fatal termination, by the occurrence of erysipelatous bronchitis and pulmonary congestion, I have already spoken of, and illustrated by the case of Edward Gresley (Case XLIV, p. 130). In a third class of cases the patients die delirious or comatose. When this occurs, some authors would tell you that the erysipelas has induced inflammation of the membranes of the brain. These notions are now, however, almost entirely exploded, and there is ample evidence, that if death takes place while the patient is in either of these conditions, the cerebral meninges are found, upon post-mortem examination, to all appearance perfectly healthy, nor can any of the products of an inflammatory process be detected; or, if there be anything amiss with them, it is that the vessels of the pia-mater contain rather less blood than they ought to do. These formidable symptoms are rather to be attributed to the circulation of a noxious blood through the brain, and to a consequent defective and depraved nutrition of that organ, than to inflammation and its products.

Let me repeat here that the amount of congestion of the vessels of the brain is very much influenced by the mode of dying of the patient. A prolonged moribund state, with heavy breathing, and especially with convulsions, increases congestion by retarding the flow through the veins; a rapid death, with much exhaustion, and faintness, what I have repeatedly remarked to you, leaves the vessels with very little blood.

I shall conclude with some remarks upon the treatment of erysipelas generally; and with especial reference to this subject, I must beg your attention to the following classification of cases, based on a due consideration of the clinical history of the disease, and the issue of different examples.

I think we may fairly arrange the various cases of erysipelas into the five following classes:—

1. There are certain cases of erysipelas which get well of themselves, and these are generally examples of the disease in a slight form, affecting the head and face only, or some other limited portion of the skin. Usually in the course of two or three days, especially if they are kept in a comfortable place, and have a little beef tea or other light nourishment, patients suffering from attacks of this intensity recover, and this, too, in some cases, despite of a certain amount of lowering treatment, and of the use of such remedies as tartar emetic.

2. In a second series of cases of this disease, the very opposite of the first, the patient dies downright, if I may use the expression: he sinks rapidly, do what you will. This mode of termination is common to erysipelas, with most of the other diseases which are due to the influence of a poison, whether it be generated in the human body, or be of atmospheric origin. Thus, in a considerable proportion of cases of cholera, it is perfectly in vain to attach very much importance to anything in the way of treatment, for the patient is dead almost before the case comes fairly under observation; and, in such instances, I doubt that any plan

of treatment ever will avail, because the morbid phenomena are of such rapid accession, almost as rapid as if the patient had taken a very large dose of arsenic or of prussic acid. The same also occasionally happens in typhus fever, the patient being killed within a very few days, or even hours, of the commencement of the attack; and so likewise, in all the exanthemata,—small-pox, measles, scarlet fever, &c.,—all treatment is often utterly unavailing, and death occurs during the first twenty-four or forty-eight hours of the illness.

Hence, then, it becomes necessary, in all endeavors to estimate the value of any particular plan of treatment in erysipelas, carefully to exclude from the data upon which conclusions are to be founded these two classes of cases: first, those which would get well of themselves; and, second, those which defy every attempt at treatment.

3. The third class comprises those which recover under a suitable treatment, but in which there is a marked tendency to death, and which there is good reason to believe would terminate fatally if left to themselves.

4. The fourth group consists of those cases which pass through the early stages of the malady more or less favorably, but which then exhibit the secondary phenomena of the disease. In this class complete recovery may take place, or death may result from the exhaustion which is frequently induced by the extension of the suppurative process, and by its duration.

5. In the fifth and last class, not only do the secondary phenomena of the disease manifest themselves, but, by some means a morbid material finds its way into the circulation, and as a result of this, formations of pus take place in various parts of the body, and the patient dies of purulent infection, or pyæmia, as it is called.

The treatment for erysipelas which I have for many years past adopted, is the supporting plan; and this I would, from a long experience, recommend to you, under the conviction that it is the best adapted to save life, and check the progress of the disease; and that under it, if begun early and with decision, you will seldom have to deal with the secondary phenomena of the lady.

treatment consists in the free administration of food and such as beef tea, and some form of alcohol—brandy by preference—in precisely regulated quantities, at stated and short intervals; if drugs are needed, ammonia, bark, and chloric ether, in forms most agreeable or least offensive to the stomach, may be given. The beef tea, or other animal broth or soup, and brandy, should be given at stated times, in small doses, two or three ounces of the former, and from two drachms to half an ounce or an ounce of the latter slightly diluted with water. Two different forms of alcoholic fluid should not be given at the same time, such as wine and brandy, or beer and brandy, or gin and brandy; and for other nourishment it is desirable to observe the same rule, as far as possible. You must attend closely to the digestive power of your patient, and be careful to avoid exciting dyspeptic symptoms, such as nausea, sickness, hiccough, flatulence, by giving too much at one time, or by too great a variety of stimulants or food.

Sometimes in the course of an attack of erysipelas, the patient may become delirious, or he may fall into a state of coma. It is during the first fourteen days of the illness, that these formidable symptoms are most apt to occur; hence the necessity of beginning early, from the first, with support and stimulants, which you will find preventive both of delirium and coma. The lower you keep your patient, the greater will be the tendency to delirium or coma, and the more violent and profound will either be, and the development of either is an indication for pressing the treatment in the same or greater doses. Sometimes you will find that the coma persists, notwithstanding all the support you can give; and then you may generally conclude with certainty that the blood has become poisoned by pus, or some other morbid agent, and that death from pyæmia is about to occur, or that local formations of pus are about to be developed in various parts of the body.

In those cases in which the disease responds to the stimulating treatment, the delirium subsides, and speedily altogether disappears; the redness and swelling diminish; the pulse becomes softer, fuller, and less frequent; the fever decreases, and the state of convalescence is rapidly established.

Sometimes, through feeble powers of digestion in the patient, or injudicious zeal on the part of the attendants, you may find that you are over-stimulating. What are the indications of this? They show themselves in sickness, in flatulence, in a sense of oppression, perhaps also in derangement of bowels. When such symptoms occur, nothing can be easier than to suspend the treatment for a few hours, to give only a little cold water, and afterwards to resume it cautiously in diminished quantities.

Case XLVIII.[1] (Vol. xlvii, p. 13.) This case serves as a good illustration of the effects of over-stimulation. Ellen Sutcliffe, aged thirty-four, admitted into King's College Hospital, on April 25th, 1855, with a very severe attack of erysipelas of the head and face. Pulse 112; respirations 28. This patient was at once ordered plenty of beef tea, and the following draught every hour: Ammon. carb. gr. ij., sp. ether. chlor. ♏x., sp. vini gallici ʒiv., aquæ ℥ij. For the three next days the erysipelatous inflammation gradually spread, and the pulse maintained its frequency, and on the 29th the draught was ordered to be given every half hour. On the 30th, however, retching and vomiting manifested themselves, and the patient became extremely prostrate, and it appeared as though the vomiting had arisen from the excessive amount of stimulants, or, perhaps, rather of ammonia and chloric ether, for the brandy had been mixed with the physic, in order to insure its being administered. She was now, therefore, ordered simply half an ounce of brandy every half hour, and a little bark and ammonia in effervescence every third hour; and from this time the case proceeded in the most favorable way. The stimulants were gradually diminished as the pulse fell, and the erysipelas subsided; and, without the occurrence of any secondary abscess, this patient was discharged from the hospital quite well, after a stay of little more than three weeks.

The following case may serve to warn you of the danger likely to arise from delaying the administration of nourishment and stimulants until the worst symptoms have showed themselves. When this patient was first admitted, his pulse was only 88; but twenty-four hours afterwards it was 112, and he was hopelessly prostrate: the time for treatment was then well-nigh passed. Had that day been spent in the steady employment of the treatment I have recommended, I think it very probable that the patient, who was a favorable subject for the treatment of acute disease, might have been saved.

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[1] This case was recorded by Mr. Hardwich.

Case XLIX. (Vol. xxxi, B. p. 36.) J. Howard Collins, æt. 27, of temperate habits and general good health, was admitted with erysipelas of the face, May 3d, 1850. He stated that he had suffered from a similar attack two years previously; that the present one had commenced, five days before, with a small boil on his nose, which rapidly increased in size; at the same time he suffered from shivering, which he attributed to exposure to cold. The inflammation rapidly extended from his nose over the greater part of his face, which was of a dark rose red when he came to the hospital. He then complained of loss of appetite and sleep, headache, thirst, and pain in his right side; his pulse was then only 88. A little carbonate of ammonia formed the whole treatment at first.

By the 4th, the inflammation had extended to the scalp, the pulse became feeble and rapid—112, and the prostration great. His nose was of a livid hue and covered with minute pustules, and it was thought advisable to make a small incision into it. Two drachms of brandy were given every half hour, and half a drachm of chloric ether with each dose of the carbonate of ammonia every four hours. In the afternoon the brandy was doubled, and fifteen minims of Battley's liquor cinchonæ given every two hours. The prostration, however, increased, and difficult respiration came on; the brandy was again doubled, an ounce being given every half hour; but even this active treatment was utterly powerless against the state of hopeless exhaustion into which he had so rapidly fallen. The difficulty of breathing increased, a cold perspiration broke out, and he died early on the second day after admission.

Almost the only morbid condition found at the post-mortem, was the evidence of recent pleurisy on the right side. The transverse colon lay in front of the liver, and had made a depression on the left lobe.

The upshot, then, of all I have to tell with respect to the treatment of erysipelas, is to give stimulants and nourishing food freely, and from the very commencement of the attack. Don't trouble yourselves with too much attention to the secretions, as some are apt to do, who imagine that the alteration of these by gray-powder, black draught, *et hoc genus omne*, is necessary to the favorable issue of the case, but who, by the time they have got the secretions into what they conceive to be a correct condition, find that their patient is fairly slipping through their fingers, and is dying, worn out and exhausted. As soon as you are satisfied that the patient to whom you are called is laboring under erysipelas, at once begin to administer stimulants and nourishing food, using the precautions I have mentioned; and what I wish above all things to impress upon you is, that this stimulating treatment should be employed from the very beginning of the attack. With respect to the bowels, you must be guided by circumstances; if they are confined, you may open them by an enema, or by a dose of castor oil or some other medicine, which will neither irritate the mucous membrane of the alimentary canal, nor exhaust the patient's strength; always keeping in view that the poison of erysipelas is exceedingly depressing in its action, and that the object of all your treatment should be, first, to antagonize the poison, and, secondly, to uphold the patient's powers, to enable him to bear up against one of the most lowering and debilitating diseases to which the human frame is liable.

Now of all the stimulants, I believe, as I have already said, the alcoholic are the best, and I have witnessed such remarkable effects, in such a variety of cases, produced by their free exhibition, that I am inclined to consider them as *antidotes* to the erysipelatous poison. If I were to be restricted to any one remedy in the treatment of this disease, I should, assuredly, choose brandy. With a commissariat well supplied with brandy, and simple means to keep the bowels open, I think I could engage to keep erysipelas at a minimum among the wounded in our army in the Crimea.

Some attach great importance to the use of the tincture of sesquichloride of iron in this disease. I have no doubt many cases, such as those which I have placed in my first group, will get well under that drug, partly and mainly because it excludes depressing treatment, partly, perhaps, from some tonic power in the medicine; but I would as soon think of trusting to it in the treatment of the third or fourth group of cases, as I would to the billionth of a grain of aconite, or arnica, or sulphur, or any other homœopathic absurdity. The remedy, so far as I know, is unobjectionable in itself, but its power to do good is small; and if you try it, let me advise you not to trust to it alone, but merely to use it as an adjunct to the treatment which I have endeavored to impress upon you to-day. For, as I before said, there is a large class of cases of erysipelas which will get well without any treatment whatever, and, indeed, in spite of depressing treatment, either because the dose of the poison which these patients have imbibed has been very small, or because their powers of resisting acute diseases are very great. In such cases you may, if you like, amuse yourselves with giving a remedy of the nature of sesquichloride of iron. But in all severe examples of the malady, place your trust in food and brandy, freely given under careful regulation, and adopted from the very commencement of the attack.

The following cases are worthy of your attention as good illustrations of the effects of the treatment I have recommended in this lecture.

CASE L.[1] (Vol. xlviii, p. 80.) Thomas Lane, aged fifty-three, a coppersmith, of intemperate habits, admitted into King's College Hospital on June 1st, 1855, in a state of violent delirium, with erysipelatous inflammation of the right cheek, and with signs of slight suppuration in the right upper eyelid. It appeared that this man had been formerly in the habit of working in distilleries, where he was constantly exposed to the fumes of spirits, and to counteract the intoxicating effects of the inhalation of these, he used to drink a very large quantity of beer; but that he had latterly changed his occupation for that of a plumber, and while following this employment he had been continually exposed to wet from the escape of water from pipes. The attack of erysipelas, for which he was brought to the hospital, commenced about a fortnight prior to his admission, with pain, redness, and swelling over the right eye, which symptoms gradually increased in severity until he was brought under observation on June 1st. By this time delirium had manifested itself, and he was so violent, and created so much disturbance, that it was found necessary to place him in a ward apart from the other patients. Pulse 86; respirations 30. The treatment upon which he was put consisted in the free supply of beef tea, milk, and arrow-root, together with half an ounce of brandy every two hours, and twenty drops of laudanum every four hours. The following day an incision was made into the right eyelid, and a small quantity of pus escaped, and the delirium and other symptoms still continuing unabated, the brandy was increased to half an ounce every hour, and bark, chloric ether, and ammonia in effervescence, were substituted for the laudanum. Pulse 76; respirations 24. The day succeeding this, the 3d, the delirium had considerably diminished. Pulse 74; respirations 22. On the 4th, the pulse was 74, and the respirations 20; and on the 5th, the erysipelas was rapidly disappearing, the delirium had entirely ceased, the pulse was 56, and the respirations 22. The stimulants were now diminished gradually from day to day, meat and porter being substituted for brandy and beef tea, and on the ninth the patient was convalescent.

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[1] From the record of the case by my clinical clerk, Mr. Wharton Hood.

Case LI.[1] (Vol. xlviii, p. 54.) J. Scarfe, aged forty-seven, a relieving officer, of temperate habits and good general health, was admitted into King's College Hospital, on May 17th, 1855, with a severe attack of erysipelas of the face and head. Pulse 110; respirations 38. He was at once put upon half-ounce doses of brandy every two hours, with a plentiful supply of beef tea, &c. A week previous to this man's admission to the hospital, he was seized with shivering and vomiting, and this was speedily followed by redness and swelling of the nose. The erysipelatous inflammation rapidly spread, and involved the whole of the face and temples, the eyes being completely closed. The day following his admission, the pulse was 104, and the respirations 42; and the day after this, the 19th, the report of him is this:—"He is rather worse to-day: pulse 112; respirations 38: he had no sleep last night, and is now very restless, and wanders occasionally: tongue thickly coated with a brown fur: the swelling of the face is slightly diminished." The brandy was now increased to half an ounce every hour, and on the 20th, the pulse had fallen to 96; and on the 21st, this report is entered in the book:—"The redness and swelling have entirely subsided, except under the right eye: pulse 88; respirations 35: he sleeps well; the tongue is cleaning, and he is asking for meat." On the 22d, the brandy was diminished to half an ounce every two hours, and the convalescence was now rapidly established, the only indication of suppuration, anywhere, being the existence of a very small collection of pus in the right upper lid, which was opened on the 23d. On the 26th, all brandy was withdrawn, and the patient put on porter and quinine; and a few days subsequently he was discharged quite well.

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[1] Reported by Mr. Wharton Hood.

Case LII.[1] (Vol. xlviii, p. 60.) Martha Tasker, aged 21, a healthy-looking girl, with a marked hysterical countenance, admitted into the hospital, on May 23d, 1855, for pain in her left side,—which appeared to be hysterical,—and occasional headache. For this she was put on steel and a pretty liberal diet, and on June 8th, was ordered to leave off all medicine, as she was reported to be "nearly well." Three days after this, however, it was found that she was attacked with erysipelatous inflammation in the right eye and side of the face, which was red and swollen. She now complained much of pain in the head and giddiness, and she was at once put upon half-ounce doses of brandy every third hour. It almost appeared as though this treatment put a stop to the progress of the disease, for the erysipelas ceased to spread, and in three or four days the face was almost well. On the 14th, however, the right arm became the seat of erysipelatous inflammation, which, treated in a similar way, rapidly disappeared.

Case LIII. M. E., aged 22, a needle-woman, greatly overworked, was admitted into King's College Hospital, on May 9th, 1855, with a severe attack of erysipelas, involving the whole of the face, which had commenced four days prior to her admission into the hospital. It appeared that this patient had had two previous attacks of the disease, one nine and another six years before the present.

On admission, her pulse was 116, and her respirations 26, and she was very restless and quite unable to sleep. Plentiful supplies of beef tea were ordered her, together with half an ounce of brandy every hour. On the 11th, the pulse was 120, and the respirations 30; and on the 12th, the erysipelas exhibited a tendency to spread to the scalp, but the pulse had fallen to 118, and the respiration to 22.

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[1] Reported by Mr. Wharton Hood.

On the 14th, an immense improvement had taken place: the swelling had very much diminished, desquamation was beginning to be established, and the pulse had come down to 89, while the respirations were 22. On the 15th, the report is as follows:—"The swelling has almost entirely subsided; the cuticle is peeling; the pain in the head is quite gone; the pulse 87; respirations 22."

The stimulants were now gradually diminished; and on the 16th the pulse was 72, and the respirations 20; on the 17th, pulse 66, respirations 18; and on the 18th she was fairly convalescent, the pulse being 62, and the respirations 16, while the only appearance of a secondary abscess was a small pustule on the margin of the left upper eyelid. Full diet, porter, and quinine were now gradually substituted for the brandy and beef tea; and the patient was discharged quite well on May 26th, 1855.

CASE LIV.[1] (Vol. xliv, p. 215.) Elizabeth Knight, aged 56, a married woman who had had fourteen children, was brought into King's College Hospital, on March 15th, 1855, with a curious chorea-like shaking of the right arm, and erysipelatous inflammation of the right cheek. About four weeks previous to her admission to the hospital, she suffered a great fright from waking up one morning and finding a niece who was sleeping with her, lying dead by her side, and a few hours afterwards she was seized with chorea-like jerkings of the right arm, which have continued ever since, but which cease entirely during sleep. From that time she remained in a very weak and low state, fainting occasionally, until March 11th, when she was taken with pain, redness, and swelling in the right cheek, and violent shivering; and on the 15th she was brought to the hospital. Her pulse was then 68, exceedingly weak, and her respirations 48, loud rhonchus being audible all over the chest. The treatment to which she was subjected consisted in the exhibition of half an ounce of brandy every two hours, five grains of carbonate of ammonia every four hours, with an opiate draught at night, together with free supplies of beef tea. On the 16th, the erysipelas was evidently spreading, the pulse was 86, and the respirations 46; the brandy was now increased to half an ounce every half-hour. On the 17th, the whole of the right side of the face was involved, and violent retching had come on, all the brandy taken being almost immediately returned. The brandy was therefore at once left off, and an ounce of wine was given her in an effervescing water every half-hour. On the 19th, though the erysipelas had involved the whole of the face, yet the patient appeared decidedly better, the convulsive movements of the arm and the vomiting having entirely ceased. On the 20th, the erysipelatous inflammation was rapidly subsiding, and desquamation of the cuticle was commencing. The pulse had now fallen to 94, and the respirations to 20. From this time, the patient gradually improved, the pulse coming down a few beats daily, while, as in the other cases, the stimulants were by degrees lessened and lessened, until, on the 30th, all erysipelas had gone, and the patient was put upon porter, quinine, and a generous diet. The only suppuration which occurred in this case consisted of a little pustule on the edge of the right lower lid, and a small abscess in the right armpit, both of which speedily got well. The convulsive movements of the arm never returned, while this patient was under observation, after their first disappearance on the 19th.

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[1] From the notes of my clinical clerk, Mr. Dunn.

CASE LV.[1] (Vol. xxi, p. 168.) James Webster, a temperate man, but past middle age, and therefore not the most favorable subject for acute disease, was brought into the hospital on the 23d of October, 1847, in a state of high delirium and quite unconscious, apparently suffering from severe erysipelas of his head and face, which were red and covered with a branny scurf; there was also a large slough over the sacrum.

For some time after his admission he appeared in a dying state, and passed his evacuations unconsciously. He was ordered a full dose of opium, strong beef tea, half an ounce of wine every two hours, and carbonate of ammonia; under this treatment he rallied, and partially recovered his consciousness by the afternoon of the 24th, but could remember nothing about the commencement of his illness.

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[1] Reported by Mr. Lakin.

He continued for some days in a drowsy, wandering state, but could be roused, and then answered questions rationally. There were very considerable muscular tremors, such as we see in bad fever cases and drunkards. His pulse was 108, small and feeble. An equal quantity of brandy was ordered in place of the wine, and he was put on a water bed.

On the 28th, he was much better,—quite conscious and rational: pulse 96, but still very feeble: the redness had quite disappeared: the slough on the sacrum seemed alone to prevent a rapid recovery. On Nov. 3d, he was ordered a mixture of quina and sulphuric acid; also a pint of porter and six ounces of wine instead of the brandy. On the 10th, the slough separated. On the 18th, he was much better; his tongue clean and appetite good. His convalescence was rather slow; but taking into consideration his age and the severity of the attack, it was, on the whole, most favorable, and he was discharged, well, on the 18th of December.

Case LVI.[1] (Vol. xxxi, B. p. 121.) John Jones, æt. 44, a man of intemperate habits, after a fall while intoxicated, was attacked with erysipelas of the face, for which he was admitted into the hospital, April 27th, 1850.

His face was then much swollen, and of a deep red hue from the inflammation. The usual constitutional symptoms were present,—hot and dry skin, thirst, loss of appetite, pulse 84. He was ordered a quart of strong beef tea, a small quantity of brandy and chloric ether. The brandy was increased the next day to half an ounce every two hours. On the 30th, he became very delirious and would not stay in bed; his pulse was feeble and had risen to 100; the brandy was increased to half an ounce every hour. By the following day, the erysipelas had somewhat extended, but the delirium abated, and his pulse began to decline. On the 2d of May, the pulse was 76; and on the 4th, 72; and the brandy was reduced to half an ounce every two hours.

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[1] Reported by Mr. J. C. Dickinson.

He continued improving, and his appetite returned, but the inflammatory process had run on to suppuration, and an abscess began to point in his cheek, which discharged itself on the 14th. By the 18th, he was quite well, but rather weak, and left the hospital.

# LECTURE VII.[1]

## *On Erysipelas of the Fauces.*

GENTLEMEN,—When speaking in a former lecture on the subject of erysipelas, you may remember that I mentioned, cursorily, a form of inflammation which I believed to be erysipelatous, and which, commencing in the throat, confines itself entirely to the faucial region. This affection is not a common one, and, so far as I know, has escaped the notice of systematic writers :[2] at the same time you will do well to pay particular attention to the peculiar characters manifested by it, as instances may occasionally come under your own notice, and the disease being of a very fatal tendency if not at once treated with great decision, it is of the utmost importance that you should immediately recognize it. The cases are, if I may so speak, of the kill and cure class: the symptoms last but a short time, and the disease runs its course, whether for life or death, in a brief period. If the treatment be vacillating, you will probably lose your patient; but if you have decided upon the right line, and pursue it promptly and steadily, you need scarcely ever lose a case. For these reasons, gentlemen, I shall beg your attention to-day to the particulars of some extremely interesting examples of this affection, one of which is still in the hospital: but first let me give you, very briefly, a connected outline of the characteristic features of the malady, and of the treatment I have found effective for its cure.

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[1] This lecture is made up of two, delivered at King's College Hospital; the first in May, 1851, the second in November, 1852. The first was published in the *Medical Times and Gazette* of June 5th, 1852; the second in that of July 15th, 1854.

[2] Dr. Elliotson, indeed, in his *Lectures on the Principles and Practice of Medicine* (Lond. 1839), speaks, at page 371, of erysipelas affecting the throat as follows, from which it seems evident that the affection I describe was not unknown to him:—"Erysipelas is a disease which is by no means confined to the surface of the body. You will continually see the throat affected. If the inner part of the throat and mouth are the seat of disease, you will see the throat red; the tongue red; the mouth complained of by the patient as exceedingly hot; perhaps a short cough, and a difficulty of swallowing. In fact, there is a sore throat. Very frequently, too, it will run down the membrane lining the tubes; so that you have a very great cough, and a difficulty of breathing. You have more or less bronchitis; and sometimes there is really *severe* bronchitis; but for the most part, it is only a *superficial* sort of inflammation—erysipelas of the mucous membrane; and will go away without the adoption of any strong measures." A slight examination of the cases which form the subject of this lecture will, I think, nevertheless convince the reader that they differ widely in the intensity of the local and general symptoms, although not in their essential nature from those here referred to. In my lecture on erysipelas, I mentioned that the idiopathic form of that disease very frequently commences with redness and soreness of the throat, and from thence the inflammation spreads externally, and I gave an instance (Case XLII, p. 191) in which the throat affection was throughout the most prominent feature: I think it must be to such cases as these that Dr. Elliotson alludes.

The peculiarity, then, of the disease consists in this:—that the force of the poison seems to fall upon the pharynx and to paralyze it; and it must do this, either by benumbing the sensitive nerves, through which the muscular contractions are usually excited by the contact of food, or by extending to the muscles themselves and paralyzing them directly, or, it may be, in both these ways. If you look into the throat of a patient laboring under this affection, you will find the pharyngeal mucous membrane exhibiting a peculiar dusky-red color, the fauces will be perfectly open, and you will be unable to discover any mechanical impediment to free deglutition; and if, now, with your finger, or a pen or probe, you touch the back of the pharynx, you will find that none of the pharyngeal muscles are thrown into action, as they invariably are in a state of health; in other words, you cannot excite the reflex actions necessary for deglutition; and if you give the patient something to swallow, as soon as he gets the liquid or solid, whichever it be, upon the back of the tongue, instead of its being grasped by the contraction of the muscles of deglutition, and guided, as it were, into the œsophagus, in consequence of the complete palsy of these muscles, it falls by its own gravity into the larynx, and is thence immediately ejected, by a powerful expulsive effort, through the mouth and nostrils.

I have seen, and have notes of several of these cases which have occurred both in hospital and in private practice, and all those which were treated upon the antiphlogistic plan died; but for some years past now, from being better acquain true pathology of the malady, I have invariably resorted to an opposite mode of treatment,—an actively supporting and stimulating one, precisely that, in fact, which you have heard me advocate as most beneficial in œdema glottidis—except that we can get on here without calling in the aid of the surgeon—and since I have pursued this practice, I have scarcely met with an instance of this affection which has not recovered.

In these cases, the patient is apt to die, not from want of air, as when the erysipelatous poison attacks the mucous membrane of the larynx, but from want of food, the inability to swallow being even greater than in cynanche tonsillaris, which creates a mechanical obstacle. The difficulty therefore in the treatment is to get sufficient nourishment into the patient; but if a person cannot gain admittance into a house through the front door, the first thing to which he would most probably resort, if it were very important that he should gain entrance at all, would be to try at the back door. So here, since there is an almost insurmountable difficulty in introducing food into the stomach through the mouth, the only resource left us is to throw beef tea injections, containing large doses of quinine, into the rectum, and feed the patient in this way. Then the back of the fauces should be lightly touched with the solid nitrate of silver, or freely washed with a strong solution of it, and as soon as the power of swallowing begins to return, which it generally does, under this plan of treatment, in the course of from twenty-four to forty-eight hours, frequent and large doses of brandy, ammonia, chloric ether, and beef tea should be exhibited by the mouth. If, however, from any cause, the plan of feeding by the rectum fails to restore the power of deglutition, you must then have recourse to feeding by the stomach-tube; but this mode of proceeding is generally unsatisfactory, and must only be resorted to when all the other means, which I have just mentioned, have been fairly tried and found unsuccessful. Very patient spoon-feeding, carrying the food as low down in the fauces as possible, will often be found successful. A skilful and practised nurse will succeed in this where a more scientific person would fail.

CASE LVII.[1] (Vol. xxxiii, p. 191.) The first case I shall speak of is that of the man, George King, in Sutherland ward, aged sixty-four; he is tall, and of spare build, and he looks much older than the age he gives, and appears as if he had been suffering for some time from illness. This condition, he tells us, resulted from a severe attack of rheumatic fever, under which he suffered about a year and a half ago, and which was accompanied with cardiac affection.

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[1] The record of this case was kept by Dr. Bridgwater.

Since that attack, he has been more or less an invalid, from a succession of catarrhal affections, up to Friday, the 25th of April, 1851, on which day, while he was crossing St. James's Park, under a sharp northeast wind, he was suddenly seized with a sensation as if something had forcibly closed his jaws, and he found, to his alarm, that he could not open his mouth with the most violent effort. The affection at the same time seemed to attack the throat, and completely to prevent his swallowing. When he arrived at Pimlico he could not eat his dinner, and was unable to swallow some gin and water which he attempted to take. He could get it into his mouth, and it even reached the pharynx, but would go no further; it seemed, to use his own expression, "to stick in his throat," and was soon returned. He made frequent and ineffectual attempts to swallow, but, from this time till the following Tuesday, he remained without taking a particle of nourishment.

When he was admitted into the hospital, attempts were made to examine his throat, but, in consequence of the state of trismus which prevailed, the teeth could not be separated sufficiently far apart to admit of any examination being made. I will read you the following extract from notes made at the time of his admission by Dr. Salter, who then ably filled the office of house-physician: "On attempting to look into his mouth, I found that I could not separate his jaws more than about one-eighth of an inch,—just a chink. I gave him some water, which he attempted to swallow. He performed the buccal and lingual, and, to a certain extent, the pharyngeal part of deglutition, but then, with a spasmodic effort to get it further, he choked, his eyes looked as if they would dart from his head, and up it came. Repeated attempts merely led to repeated failures. I could neither look into his throat, nor insert my finger to feel it."

At my own visit, in the afternoon of the same day, just the same state of things continued to exist: deglutition was quite impossible, the attempt to swallow was followed by the forcible ejection of the food through the mouth, and in some degree through the nostrils, by the agency of a spasmodic expiratory effort.

In considering what might be the affection calculated to give rise to these symptoms, I first thought of stricture of the œsophagus; but, upon inquiry into the history of the case, it seemed very unlikely that the difficulty of deglutition could arise from this cause. The affection came on suddenly, which is not the case in ordinary stricture of the œsophagus. Stricture of the œsophagus generally results from cicatrization of ulcers and contraction of the tissues adjacent, or from some malignant formation round the tube, and such strictures are most apt to occur in the lower part of the tube not far from the cardiac orifice. Did such a stricture exist, the dysphagia would have been of a different kind. Here matters were suddenly and forcibly rejected, while in the former condition the food is swallowed to a certain point, and seems to encounter an obstacle at a certain situation, to which the patient points with his finger; then it is either stopped completely, and afterwards ejected, but not with the force and rapidity which we observed in this case, or by a considerable effort it is made to overcome the obstacle. A spasmodic state of the œsophagus might create a dysphagia like that noticed in this case, but the symptoms would scarcely have come on so rapidly, nor would the danger of choking have been so imminent as in our patient. It seemed to me, that the seat of obstruction or difficulty was not so low as the œsophagus, but rather in the pharynx, and that the food encountered it immediately on passing from the mouth.

Difficulty of deglutition sometimes results from an affection of the medulla oblongata; in such cases, the dysphagia does not come on suddenly, and is not accompanied by the acute symptoms present in this case. The attempt at deglutition is in both cases quite alike, and its failure is from a similar cause, namely, spasm of the glottis; but in the one the affection is acute, in the other it is chronic.

Aneurisms, again, frequently give rise to dysphagia: difficulty of deglutition is often a most important diagnostic mark of thoracic aneurism; but this, of course, comes on gradually, as the dimensions of the aneurismal tumor increase, and the dysphagia is by no means of the complete kind which occurs in the affection under our consideration.

Another cause to which we might refer these symptoms is, inflammation of the epiglottis, which is capable of producing great difficulty of deglutition ; and, if we bear in mind the position of the epiglottis, we shall not be at a loss to conceive how this takes place. If the epiglottis be enlarged, it lies as a tumor between the rima glottidis and the base of the tongue, and would not only offer some degree of obstruction to the passage of the food down the pharynx, but it would prevent that complete apposition of the root of the tongue to the rima glottidis which is necessary for the perfect closure of that chink. When the epiglottis is diseased, the difficulty of swallowing arises from the tendency of the food to pass into the larynx, the effect of which is, a violent spasmodic action of all the muscles of the part, and forcible projection of the food upwards into the posterior nares. Thus this peculiar mode of regurgitation of the food becomes a most valuable point in the diagnosis of a diseased state of the epiglottis; for you will always find, that where difficulty of deglutition arises from an inflamed or ulcerated state of the epiglottis, the food is thrown upwards into the nose, and is frequently forced out of the nostrils with considerable violence. The dysphagia in both these cases is very similar, although not exactly alike; in our case, the food was chiefly rejected through the mouth; only a small portion of it passed through the nostrils. Moreover, in disease of the epiglottis, there is more or less affection of the voice; in our patient that function was not impaired. But conclusive evidence is obtained from feeling the epiglottis with the finger; in our patient it was soft and flexible; in inflammation it would have been swollen and stiff. Hence I was led to exclude epiglottidean disease from my diagnosis.

Again, it might have been a case of common cynanche tonsillaris. If we could have opened his mouth, this point might have been decided at once; but the complication of trismus hindered our efforts at diagnosis very much. However, I decided against cynanche tonsillaris upon the suddenness and force of the rejection of the food. In cynanche, the deglutition is extremely difficult and painful, and made with great effort and suffering; but it is not impossible, nor is rejection of the food a constant symptom, and when it does occur, it is not made in that violent sudden way which we observed in this case, nor with the signs of choking.

If, then, there was no cynanche, no stricture, no laryngeal or epiglottidean disease, no aneurismal or cerebral affection, what could have caused this remarkable dysphagia?

When I had examined the patient, some other cases were brought forcibly before my mind, and I felt certain that the peculiar symptoms in the present instance resembled those which I had observed on previous occasions with great interest.

CASE LVIII. The first occurred to me many years ago in private practice. I was called to see a lady who had for some days been suffering from influenza. The principal affection, however, was great difficulty of deglutition. When she took any food, it evidently reached the isthmus faucium, but there it seemed to excite choking in consequence of a portion apparently passing into the larynx, whence it was repelled with considerable force; so urgent was this difficulty of swallowing, that it was impossible to give her food, for fear of producing suffocation. When I looked into her throat, I saw that there was no mechanical impediment to the passage of the food into the pharynx; and all that I could observe was slight redness of the velum, and a dusky hue of the mucous membrane of the pharynx. I found, also, that no amount of stimulation of the mucous membrane of the velum would excite contraction of the palatine or pharyngeal muscles. Believing that the phenomena depended upon an inflamed state of the mucous membrane of the throat, and not having yet learned that the proper antiphlogistic in such cases is support and nourishment, I was content to trust in the application of blisters and leeches to the throat. But my patient became speedily more exhausted and rapidly sank.

CASE LIX. The second case occurred in the person of a middle-aged woman, the wife of a respectable tradesman in Westminster. I had no difficulty in immediately recognizing the similarity to the former case; the symptoms were just the same: if fluids were given, they ran down into the larynx, and were expelled with cough; and any mechanical stimulus failed to excite contraction of the velum. There was dusky redness of the velum and pillars of the palate. Profiting by former experience, I determined to exclude all depressing treatment in this case. I ordered injections of strong beef tea with ten grains of quinine to be given every four hours, and I freely applied the solid nitrate of silver to the fauces. The injections were given regularly, and the next morning I found my patient much better: she could swallow a little, and appeared stronger. I now ordered her to take beef tea and wine cautiously by the mouth, in small quantities at a time, and the quinine was also given by the mouth. In less than forty-eight hours more she had completely recovered.

CASE LX. The third case was that of a gentleman of fortune, whom I attended at one of the hotels in my neighborhood, in the month of March, when erysipelas was rather prevalent about town. The symptoms under which he labored were very similar to those which I have just detailed in relating the other cases. There was great difficulty of deglutition, but he had some power of swallowing; there was also the same tendency for fluids to pass into the larynx; the muscles of the palate showed the same want of contractile power; the mucous membrane was of the same dusky-red hue. This patient was treated by support and stimulants, and the local application of nitrate of silver. As he was in affluent circumstances, I gave him turtle-soup and port wine, and quinine. He got quite well in a few days.

CASE LXI. Not long since I was called to see a woman who was suffering from symptoms of the same character. In this, as in the other cases it was impossible, by any amount of stimulation to excite contraction of the muscles of the soft palate; and if we administered a small quantity of fluid, it ran down into the glottis, causing violent irritation and choking. She had been ill some days, and had been treated by leeching, blisters, and mercury, as in my first case, and was in a state of extreme exhaustion when I saw her. The isthmus was quite open, and there was no impediment to deglutition except the paralytic state of the muscles. This patient died after an illness of two or three days: she died, indeed, while I was in the house.

You see that these were all very serious cases, and that I am justified in calling them kill or cure cases, so brief is the period of their duration whether for weal or woe; and, althare rare cases, you will, I am sure, appreciate the importance of being prepared for them, so that you may not be at a loss if perchance one should fall to your care. Though these are formidable cases to witness, and run their course very rapidly, terminating in death or recovery within forty-eight hours, they are, I believe, if taken soon enough, perfectly amenable to treatment, and I feel persuaded that the first case which I described would have recovered, if she had been treated on a different plan. At the time that it occurred to me, I was not familiar with the symptoms—I was not "up to the disease," if I may so express myself; and I hold the opinion, that such cases would almost always recover if subjected to proper treatment before extreme exhaustion had come on.

Now, believing that our patient up-stairs was suffering in the same way as those others whose cases have been related, I put him on the same treatment as that which I had followed in the successful cases: ten grains of quinine diffused in two or three ounces of strong beaf tea were administered every four hours in the form of an enema. I should have also ordered the nitrate of silver to be applied to the throat, but that the state of trismus prevented his mouth from being opened.

On the following day he was very much better: his mouth could be opened to the extent of half an inch, and he could swallow a little liquid.

On the 30th, he could open the mouth sufficiently to enable Dr. Salter to examine his throat; and then, although considerable progress had been made towards recovery, the mucous membrane of the upper and back part of the pharynx was found of a purplish, dirty-red color, indicating, as I thought, the existence of a low erysipelatous inflammation. There was no swelling of the mucous membrane, and no mechanical impediment to the passage of food into the pharynx, but the velum did not contract freely, as it does in health, upon the application of a mechanical stimulant.

The case of our patient King differed remarkably from the others, in being complicated with a state of trismus; which condition, however, I think, admits of explanation on the supposition that the principal source of irritation was the throat. If call to mind the presence of the extensive nervous plexus outside the tonsils and isthmus of the fauces, which is called the pharyngeal plexus, and consider how this is formed, you will not be at a loss to account for this symptom. The pharyngeal plexus, as you know, is made up of fibres from the vagus, glosso-pharyngeal, and sympathetic nerves. Irritation of the ramifications of the two former nerves may be readily propagated to the medulla oblongata, so as to affect the motor portion of the fifth nerve, which is implanted there, and by which a convulsive state of the muscles of mastication may be excited and maintained.

This man, as I have said, recovered to a slight extent his power of swallowing the day after his admission into the hospital, and was able to open his mouth slightly. We then discontinued the quinine injections, and gave him wine and nutritious food and quinine by the mouth. Under this plan, his power of deglutition was completely restored in two days; but there remained a catarrhal state of the mucous membrane of the trachea and large bronchial tubes, for which he still remains a patient in the hospital.

Now, that this affection of the pharyngeal membrane is of the erysipelatous kind, I think I am justified in affirming from the following considerations:—First, from the rapid invasion of the attack, and the great constitutional disturbance with which it is accompanied. Secondly, from the local redness; and, thirdly, from the great prostration with which the attack was rapidly followed, which was sufficient to kill two out of five cases. Our patient King, as well as all the others whose cases I related to you, seemed to succumb at once under the influence of some powerfully depressing poison, just as patients attacked with external erysipelas do. Then, it is well known, that cases of erysipelas of the head and face often commence with sore throat; or that a soreness and redness of the fauces are developed simultaneously with the appearance of the first patch on the face.

The marked difference in the treatment of the successful and of the fatal cases likewise favors the opinion, that the affection was erysipelatous in its nature. The two fatal cases were treated by a depletory and depressing plan; the three successful ones by a supporting plan, which consisted in, first, the careful avoidance of everything tending to produce fatigue, or exhaustion, or depression; secondly, in the frequent administration of large doses of quinine, with beef tea in the shape of enemata, with or without brandy, and afterwards, when the power of deglutition returned, the exhibition of bark, ammonia, wine, &c., according to the circumstances of the case; thirdly, in the local application of the nitrate of silver to the throat.

The erysipelatous character of the affection is further indicated by its extension to the trachea and bronchial tubes, giving rise to the bronchitis under which the patient subsequently suffered, and from which he is now recovering, having been subjected to a similar course of treatment to that first adopted.

The other sore throats which bear the closest analogy and resemblance to this are that from influenza, that connected with diphtheritis, and also that of scarlet fever. The last is very readily distinguished by its ulceration and sloughing character, the diphtheritic throat is characterized especially by the plastic exudation which covers it, which again you will distinguish from the aphthous throat which occurs in phthisis, in carbuncle, and after other low diseases, and which has a non-plastic exudation in which the *oidium albicans* abounds. Influenza produces a state of throat very difficult to distinguish from this which I have been describing. I cannot say, however, that I have ever seen under influenza the dysphagia go to the same extent as in the erysipelatous throat. To the eye both states of throat are alike; the mucous membrane of a dingy, dusky red, and the fauces quite open. In the influenza throat the mucous membrane has generally a more lax appearance, and the submucous glands of the velum are more prominent; the uvula is sometimes quite œdematous. In the erysipelatous throat there is more tendency to swelling in the tonsillitic region, but the swelling, according to my experience, is never such as to create a mechanical impediment to deglutition as in quinsy.

I shall conclude this lecture with the history of two other cases, both interesting examples of the disease we have been considering; one of them is especially deserving your attention, as from the case having terminated fatally, you can have before you the whole clinical history of the disease.

CASE LXII. (Vol. xxxviii, p. 77.) John Covey was admitted into King's College Hospital, Nov. 2d, 1852. He was sixty years of age, and therefore not a good subject for acute disease. From the notes of the case, we learn the following history:

His health had been pretty good until Oct. 27th, when he was seized with shivering, which was followed by fever and loss of appetite; at the same time he experienced some difficulty in swallowing. The last symptom gradually increased up to the day of his admission.

I shall read you the description of his condition on Nov. 2d, immediately after his admission, as entered in the case book: "The patient breathes with some difficulty, as if there were a collection of mucus in the larynx and trachea. He suffers a good deal of pain, increased by pressure beneath the angles of the jaw, but not much in front over the anterior surface of the larynx. There is no enlargement of the glands of the neck apparent externally. His chief complaint is of difficulty of swallowing: when he attempts to swallow anything, it seems to go the wrong way, and appears as if it would suffocate him. He can swallow a little arrow-root, but even that with considerable difficulty. When the food or the fluid which he attempts to swallow gets to the back of the tongue, instead of being guided by the action of the faucial muscles into the pharynx, it seems to fall towards the glottis, and then excites a spasmodic state, producing a feeling of suffocation, and is forcibly ejected, partly through the mouth, partly through the nose. There is no actual impediment to the passage of the food into the pharynx: the tonsils are not at all enlarged, and the pharyngeal mucous membrane looks red, but very slightly swollen, and there is a good deal of mucus upon it. When touched with the finger or spatula, the pharynx is not, as in health, thrown into action, apparently in consequence of paralysis of the pharyngeal muscles. The peculiar state of the mucous membrane extends to the larynx, for the epiglottis feels slightly swollen, and he spits up a good deal of mucus. He is very restless and sleeps badly at night. Bowels confined: pulse 96: respirations 30." Urine acid, containing blood in considerable quantity; blood corpuscles in abundance were seen under the microscope, and numerous casts of kidney tubes, apparently consisting mainly of blood-cells, as if the result of the rupture of one or more Malpighian capillaries.

You are aware that it is not uncommon to meet with slighter cases of erysipelatous inflammation of the fauces in connection with erysipelas of the head and face; that affection, indeed, not unfrequently begins in the throat and thence spreads outwards through the nose and mouth. In this case, as in the whole class to which it belongs, so far as my experience goes, no tendency of the morbid process to spread outwards has been manifested. In Covey's case, unlike the others, there was some tendency to spread to the laryngeal and bronchial membranes. Many fatal cases of œdema of the glottis originate in this way by the extension of an erysipelatous inflammation to the laryngeal mucous membrane.

Our patient was promptly treated in the manner I have recommended in this lecture: the throat was freely washed with a solution of twenty grains of the nitrate of silver in an ounce of distilled water; a mustard poultice was applied to the throat externally; he was ordered two drachms of brandy in arrow-root every three hours, and carbonate of ammonia with chloric ether was also freely given.

On the 3d, there was no improvement: prostration very great; pulse 90, and very compressible; he could scarcely swallow anything; the attempt to do so nearly suffocated him, and he had taken hardly any of the brandy and arrow-root. The mucous membrane of the throat was red, and secreting a quantity of muco-purulent fluid; the uvula was slightly swollen.

The solid nitrate of silver was now applied freely to the mucous membrane of the fauces, and he was ordered to have an enema, consisting of ten grains of quinine in three ounces of strong beef tea, every three hours, the rectum having been first cleared out by an enema of warm water. He was allowed brandy if he could swallow it.

The next day, Nov. 4th, the report was satisfactory: the pulse was better; he had had the enemata regularly, and retained them all; he was able to swallow much better, and to take at least nine-tenths of what was offered him.

On Nov. 5th, he was still improving: the throat was less sore externally; the secretion much diminished; he was able to swallow all that was brought him; pulse 80. The brandy and the enemata had been administered regularly up to that time, but the latter were then discontinued, and he was ordered chloric ether, ammonia, and bark. From this date, our patient rapidly recovered. On the 6th, his pulse had fallen to 70, and all difficulty of deglutition had disappeared; the urine assumed its natural condition; but the patient continued weak for a long time, and did not leave the hospital until the 27th.

Case LXIII.[1] (Vol. xxxiv, A. p. 50.) The last case I shall mention is that of M. A. Ayres, æt. 36, a poor married woman, who had been some time in ill health and subject to cough. She was confined with her third child on the 24th of September, 1851. There had been several cases of fever in her house; she was therefore not surprised when on the third day she was attacked with symptoms of fever,—thirst, parched mouth, hot skin. The following day these symptoms increased; her thirst became excessive, and she found herself unable to swallow.

She obtained admission to the hospital on the 30th, and was then suffering from great dyspnœa and difficulty of swallowing. On examining her throat, however, no obstacle could be observed; the interval between the pillars of the fauces was not at all contracted, but the mucous membrane looked purple, and the pharyngeal muscles refused to respond to any stimulus applied to them. When food was taken it seemed to fall upon the glottis, causing spasm and symptoms of choking.

She was ordered strong beef tea with half an ounce of brandy every hour, besides carbonate of ammonia and chloric ether.

On the 31st, her pulse and respirations were 120 and 40 respectively; they rose in the evening to 126 and 46, and the next morning they were 128 and 44.

On the 2d of September, the eighth day from her delivery, there was less difficulty in swallowing: the secretions of milk and the lochia continued. On the 3d and 4th, her breathing and power of swallowing improved still further, and she was able to eat with comparative ease. The pulse and respirations continued very high—120 and 42, and the brandy was increased to an ounce every hour.

On the night of the 6th, she was seized, while swallowing some fluid, with a convulsive attack, and gasped for breath. The dyspnœa remained for three-quarters of an hour afterwards, but was relieved by hot fomentations.

She continued, during the 8th, 9th, and 10th, in very much her former condition, with rapid pulse and breathing and a flushed face, complaining chiefly of her throat feeling parched; but she was able to swallow, breathe, and speak very fairly, and her cough was trifling. The brandy was reduced to six drachms every hour, and finally to rather less, with a little wine.

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[1] Recorded by Dr. Maurice Davis.

On the 11th, after being removed from her bed and washed, she was seized with rigors, never rallied, and died in about twenty minutes.

On making a post-mortem examination, the pharynx and larynx appeared healthy, and the pharyngeal mucous membrane which during life had a purple hue, now looked anæmic. The upper parts of both lungs were congested, the rest anæmic. The lining membrane of the uterus was purple. All other parts exhibited a remarkably anæmic appearance.

## LECTURE VIII.

### *On the Treatment of Acute Internal Inflammations.*[1]

GENTLEMEN, we have lately been watching with great interest the patient, Jane Cook, aged nineteen, who is still in the hospital, CASE LXIV.[2] (Vol. lvii, p. 17.) Her case has afforded us a good illustration of the phenomena of disease in its most acute form: she has had pericarditis in connection with rheumatic fever, some degree of endocarditis, and pneumonia with consolidation of about a fourth of the posterior part of each lung. I shall take this opportunity of making a few remarks upon the treatment of acute internal inflammations generally.

This patient is rapidly recovering, and, indeed, in an illness of unusual severity, she has had no serious drawback. On the 2d of July, rheumatic symptoms showed themselves in pain and swelling of the lower joints. On the 6th of July, a pericardial friction sound was heard over the base of the heart, which soon became distinctly audible over its whole anterior surface. On the 7th, bronchial breathing became audible at the posterior part of the lower third of the left lung, and on the 10th, the right lung was similarly affected, and to an equal extent. On the 12th, vesicular breathing began to be audible in both lungs, and the bronchial breathing to disappear.

Now this patient was treated in the manner in which (with but slight modification) I have been for some years in the habit of dealing with similar internal inflammations, especially those of the lungs and heart. Although my practice in such cases is now pretty well known, and I am proud to think is adopted by very many of my pupils in various parts of this city, and of the country, it may be useful if I take this opportunity of explaining to you the principles upon which it is based.

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[1] Delivered in July, 1857, and reprinted from Nos. I and II of the *Archives of Medicine*, edited by Dr. Beale.

[2] Reported by my clinical clerk, Mr. Mason.

And first let me describe to you in detail, as a good instance of this treatment, that to which this girl Cook has been subjected.

On admission, while yet it was uncertain how far the rheumatic symptoms would extend, she was treated with alkalies and mild saline purgatives. Bicarbonate of potass, in doses of from twenty to thirty grains, was given every four or six hours, and very soon opium was freely given, when the cardiac affection manifested itself. As much as one grain of opium was given every fourth hour. Care was taken to keep the bowels open by giving an aperient draught daily of sulphate and carbonate of magnesia. Counter-irritation was employed over the situation of the inflamed lungs by means of stupes of flannel soaked in turpentine; these were applied twice or thrice a day, and the region of the heart was freely blistered.

A principal and very important part of the treatment to which, as most of you know, I pay very special attention, is that which I may call the dietetic portion. The object of this is to support the vital powers of the patient, and to promote general nutrition, during the time when those changes are taking place in the frame which tend to check or to alter the morbid process, and to convert it into a healing process.

When a patient suffers from pneumonia, the tendency is for the lung to become solid, then for pus to be generated, and at last for the pus-infiltrated lung-structure to be broken down and dissolved. Such are the changes when matters take an unfavorable course. On the other hand, recovery takes place, either through the non-completion of the solidifying process, or by the rapid removal, either through absorption, or a process of solution and discharge, of the new material, which had made the lung solid.

It will scarcely be affirmed, even by the most ardent believer in the powers of the therapeutic art, that any of the measures which are ordinarily within our reach, such as the administration of certain drugs, or the abstraction of blood, or the application of blisters, exercise a *direct* influence in effecting these changes. Save in the case of antidotes, which directly antagonize the proximate cause of the morbid state, medicines promote the cure of acute disease by assisting and quickening some natural curative process. And he is the wisest practitioner, and will be the most successful therapeutist, who watches carefully the natural processes of cure: in other words, who studies the phenomena, both anatomical and physiological, which accompany them, and of which, indeed, they consist.

Let me, therefore, exhort you to look very carefully to this as a part of your clinical study. If you will be on the look-out, you may often meet with cases of acute disease, which recover with little or no medical treatment, and you may observe and note the clinical phenomena which they exhibit.

Allow me to anticipate your observation on this head, and to point out what you may look for in cases of pneumonia, and what you will certainly find in almost every instance.

First, the hot, often burning skin, which is so generally present in the first stages of pneumonia, will be exchanged for one bedewed with moisture, generally to the extent of free sweating.

Secondly, along with this sweating process, there will be one of increased flow of urine, and very often a free precipitate of brickdust sediment, lithate of soda, more or less deeply colored.

Thirdly, not unfrequently, expectoration will become freer, the sputa more easily discharged, they will lose their characteristic reddish, rusty color, and often become very profuse and even purulent. Now and then the purulent sputa are so abundant that it is difficult to imagine that they can have come from any other source than an abscess.

Fourthly, the chemical characters of the pneumonic sputa exhibit an interesting contrast with those of the urine. In the height of the inflammatory state, the sputa contain common salt (chloride of sodium) in abundance, and the urine is entirely devoid of it. As the inflammation becomes resolved, the salt returns to the urine and leaves the sputa.

Lastly, while all these changes are going on, the physiological functions which have been disturbed by the local malady, gradually approach their normal state. The quickened breathing, the accelerated pulse, the unnatural generation of heat, gradually subside. As all these admit of being measured by numbers, you should tabulate them in your records of cases, and you will find on each succeeding day (under such circumstances as I am now referring to) the figure assignable to each function gradually become lower until you arrive at the normal.

Now is it not plain from all this that the process of resolution of pneumonia is a distinct natural process effected by the various physical agencies which are concerned in the nutrition of the lung? A material which clogs the air-cells and minute tubes is removed, chemical changes of the most marked and obvious kind accompany the deposition and the removal of this material, and certain functions of excretion become strikingly augmented, as if for the purpose of getting rid of some obnoxious matter out of the circulation. A more exact and minute analytic chemistry than we have at present, will at some future time, beyond doubt, detect more minute changes in the blood, and determine the exact nature of the discharged matters.

One other remark I must make in connection with this subject. These acute internal inflammations are very often—I suspect always—connected with the prominence of some peculiar diathesis—the gouty or the rheumatic, for instance—sometimes the scrofulous. Of these diatheses, the main characteristic is the generation of some peculiar morbid matter which, when accumulated in quantity in this or that organ, gives rise to inflammation in it. And the determination of the morbid matter to the lung, or the pleura, to a joint or a muscle, will often depend on the direct influence of cold, or of an unwonted amount of exercise, or some mechanical injury. The evil is to be remedied by diminishing the intensity of the diathesis. This is done naturally, and is to be imitated artificially, by the elimination of the morbid element through the channels of augmented excretions, such as the sweat, the urine, and the secretions of the alimentary canal.

You will perceive, then, that my argument may be thus summed up. Internal inflammations are cured, not by the ingesta administered, nor by the egesta promoted by the drugs of the physician, but by a natural process, as distinct and definite as that process itself of abnormal nutrition to which we give the name of inflammation. Our interference either may aid, promote, and even accelerate this natural tendency to get well; or, it may very seriously impair and retard, and even altogether stop, that salutary process.

If, then, this view of the nature of the means by which inflammation is resolved in internal organs be correct, it is not unreasonable to assume that a very depressed state of vital power is unfavorable to the healing process, for how can an important vital process go on satisfactorily where there is a lack of vital power? Indeed, if you watch those cases in which nothing at all has been done, or in which nothing has been done to lower the vital powers, you will find that the mere inflammatory process itself, especially in an organ so important as the lung, depresses the strength of the patient each day more and more.

Look, indeed, at our patient, Cook, whose case has led me to this subject! See how depressed she is by the mere force of the inflammatory affection of both lungs! Her pulse small, weak, compressible, and at 120; her heart's action feeble and rapid; her surface pale and exsanguious; and yet this girl was well upheld from the commencement; she took no remedy which has any depressing influence. What must have happened in her case had she been largely bled to twelve or sixteen ounces, and taken tartar emetic freely? My impression is, that she would not have had power to go through the healing process, or that her vital powers would have been so diminished that the healing process would have stopped. The hepatized lungs would have remained hepatized (as I have often seen where blood has been freely taken), and the pericardial effusion, which did undoubtedly take place, would have been so much more considerable in quantity, as to have materially interfered with the heart's action, and added another cause of depression of vital power to those already in existence.

You will perceive, then, that according to these views, there are strong *à priori* reasons in favor of the policy of upholding our patients, even in the earliest stages of acute disease, by such food as may be best suited to their digestive organs, such as is most readily assimilated, and calls for the least effort, the smallest expenditure of vital force for its primary digestion. Nutritive matter in a state of solution—broths, soups, farinaceous matters—answers this purpose best, and also alcohol, which is directly absorbed without any previous change, and tends to feed the calorifacient process, and to diminish the waste of tissues, which would necessarily follow in order to maintain it.

Many people start with horror at the notion of giving alcohol in acute inflammatory disease. What! give brandy in inflammation of the lungs! it is only adding fuel to the fire, and cannot fail to keep up or to increase the morbid process.

Those who reason in this way take a narrow and, I must say, an incorrect view, both of the morbid process and of the healing process; they are led away by the name *inflammation*, which is likened in their imagination to an internal conflagration, to be quenched by some summary means, or to be starved out. Nothing is to be given but what is, in popular phrase, cooling; and blood, the great pabulum of animal heat, is especially to be diverted from the seat of inflammation, or to be abstracted in such ways as the peculiarities of the case will admit.

This reasoning is of the most purely fanciful kind. It rests upon a very imperfect view of the phenomena, both local and constitutional, which accompany the inflammatory process. In fact, it takes into account only two of the phenomena of this process, namely, the heat and the afflux of blood, leaving out of consideration both the exciting cause and the proximate cause of this heat and afflux of blood.

No doubt there is some analogy between an inflammation and a fire, and I might rest an argument in favor of my views upon the further prosecution of this metaphor; but I prefer to bring before you the real nature of the inflammatory process, and of the actual physiological changes which it involves.

Inflammation is a deranged nutrition. Like the normal nutrition, it involves supply and waste, and as the latter is considerable, the former will be proportionably needed. The tendency in inflammation is to the more or less rapid formation of abnormal products, such as lymph and pus; and the supplies for these formations must be drawn from the blood or from the tissues, in both cases with the effect of more or less exhaustion of vital force, in the latter with more or less extensive organic disintegration. The active chemical process which accompanies all these changes, engenders the great heat of the inflamed part.

The more this process of inflammation draws upon the blood, the greater will be the exhaustion of vital force, and the more the whole frame will suffer; the more it feeds on the tissues, the greater will be the difficulty of the reparative process. Is it not, then, important that adequate supplies should be conveyed to this process, abnormal though it be? And in what other way can the appropriate supplies be conveyed to it than through the blood, so that the waste of tissue may be stopped, and the tendency to abnormal formations be checked, at least from that direction?

And this, in truth, seems to me to be but the plain and simple fact;—you must feed inflammations as you would other active vital processes. You must, that is, feed them to prevent them from extending to, and preying on, healthy organic structures, and committing great destruction. Bear in mind, too, that you cannot stop an inflammation so long as the exciting cause of irritation is inherent in the inflamed part: you cannot cure an inflamed eye so long as the irritating particle of dust remains adherent to it. It is wise policy, then, to try and gain time, until by antidotal means, or by elimination, you can get rid of the local irritation, whatever that may be.

The physiological expression for what is commonly called *suppuration*, is a more or less rapid waste of tissue or organic matter, and a conversion of the particles so wasted into what we designate *pus*. This conversion will, within certain limits, take place in greater quantity, and the more actively, the lower the vital power of the patient. Take two cases of erysipelas, involving the same parts, and in all respects alike, and place them in adjoining beds, feed one from the beginning of the symptoms, and give him stimulants, give the other milk and beef tea; both patients will get well, but the first will have few or no secondary abscesses, the second will have them in greater or less number according as he may naturally have less or more power of vital resistance.

But to proceed to more practical points. Our patient, Jane Cook, exhibited an example of the acute inflammatory process, proceeding to a very high degree, and involving several important organs,—both lungs, the corresponding pleuræ, the pericardium, the endocardium. The tripod of life was assailed in this girl's case. It is, therefore, a highly valuable illustration of the extent to which you are likely to be called upon to proceed with the kind of treatment I have described. And on the other hand, I may remark, that being a young and healthy girl, not strumous, but clearly of rheumatic diathesis, she was as fair a subject as one is likely to meet with for the successful practice of the bleeding and lowering plan.

Yet what was our practice? Besides the drug treatment, which I have detailed at the commencement of this lecture, this girl was freely supplied with beef tea, and she had half an ounce of brandy every hour. At first the quantity of stimulant was not so great, it did not, indeed, exceed half that amount, but very soon, when we saw the inflammation spreading, and the vital power evidently diminishing, the pulse showing a marked tendency to become rapid and weak, the patient suffering from profuse sweating, which within certain limits was salutary, we did not hesitate to increase the quantity of brandy largely. You have witnessed with what result. The pneumonia subsided quickly, so that on the fifth or sixth day the signs of hepatization had disappeared, and vesicular breathing returned in each lung. The pericarditis did not disappear so quickly. On the 16th of July, the patient was troubled with diarrhœa, notwithstanding which, on the 17th, all the signs of copious effusion into the pericardium were manifest; both the pulse and the breathing were greatly increased in frequency, and the patient suffered from orthopnœa. She was freely blistered over the heart; the brandy was increased to *six drachms* every hour; beef tea was given frequently in small quantities. The opium was continued, and ammonia, with chloric ether, was also freely given.

During the spread of the pericardial inflammation the pulse rose from 104 to 120, and reached its highest point at 124. The breathing was excessively quick, but as the girl was of a highly nervous temperament, much of that rapidity was due to her extreme nervousness, which became much augmented when she was under examination: she panted rather than breathed. But it was satisfactory to find that in our daily examination of the lungs, that no cause for quick breathing existed in them at this period. It was due primarily to the cardiac disturbance, but was greatly aggravated by the hysterical state, which so often complicates, and gives a peculiar complexion to the symptoms of more serious disease in women.

It was very remarkable, that notwithstanding extensive pericarditis and some endocarditis, our patient never exhibited any marked delirium. This is uniformly the case in acute diseases, erysipelas, fever, pneumonia, rheumatic fever, in which alcohol is given, as has been done in this case. Delirium is kept off by it. This formidable complication of acute disease ceases to trouble either the patient or the physician, if the former be duly supported from the beginning. And if delirium comes on, notwithstanding that you have been giving stimulants, you will generally find it desirable to give them more freely.

This is a fact which I have so often verified, that I am enabled to enunciate it dogmatically, that alcohol, carefully administered, from an early period, in small and often repeated doses, is the best preventive of, and antidote to delirium in acute disease.

Indeed, many of you who watch my practice know how rarely that symptom gives any trouble. It is altogether the merest trifle, as compared with what I used to find it when I adopted the so-called antiphlogistic treatment. And thus a great source of danger to life is avoided.

This fact as regards the influence of alcohol in the *prevention* of delirium, is one of the most important which the clinical observation of cases, treated by stimulants, brings out. It is quite inexplicable by those who refuse to study the action and the mode of digestion of alcohol, and who, adhering to old prejudices, rest content with a practice under which, to say the least, great mortality occurs, rather than be at the trouble of carefully investigating the powers of an important remedial agent.

Another interesting point in this case deserves your attention. While our patient was getting well of the double pneumonia, pericarditis having already come on, a severe diarrhœa supervened, which depressed her very much. Did this contribute to relieve the pericarditis, as one might expect according to the ordinary antiphlogistic notions? On the contrary: immediately upon the attack of diarrhœa there ensued signs of pericardial effusion; the dyspnœa became much aggravated, and extended dulness on percussion was found to exist in a very marked manner over the cardiac region; the sounds of the heart became distant and muffled. Under the continued use of stimulants, for a short time in still larger quantity (an ounce per hour), with opium given more frequently, and free blistering of the cardiac region, these symptoms quickly subsided.

In most cases, treated as our patient Cook has been, we have found that the pulse diminishes in frequency steadily from day to day in a very remarkable way. This was not the case with Jane Cook. At first the pulse showed a disposition to fall, and it came down from 120 to 116, and remained at this point for two days; but on the occurrence of the pericarditis it rose again, but never exceeded 124. Notwithstanding the pericardial effusion it remained at this point, and afterwards fell to 120. I believe that the fall of the pulse was opposed chiefly by the highly hysterical temperament of the patient, but partly by the cardiac inflammation. The wonder was that with the extensive inflammation, and the extreme debility, the pulse did not rise more—even to 140, or higher; this, on the other hand, was obviated by the presence of alcohol, which when fully digested and acting favorably, tends to prevent the pulse from increasing in frequency, if it does not reduce it.

Lastly, our patient had a rapid convalescence. When once the diarrhœa was stopped, the pericardial effusion became quickly absorbed. The signs of effusion were at their highest point on the 17th of July; by the 20th, they had disappeared; on the 25th, the patient was fairly convalescent: just twenty-three days from her admission. From the 25th to the 2d of August, her recovery of strength and color was rapid, and she might have left the hospital at this time; but as a matter of safety, she was detained until the 15th of August, when she left quite well.

Rapidity of convalescence is not the least important feature of the cases treated by this upholding plan. When once the acute mischief is subdued, it is surprising with what rapidity the patient emerges from the invalid condition. Of this we have numerous examples in all forms of acute disease, and in none more than in pneumonia, erysipelas, and continued fever.

In conclusion, let me impress upon you, that in supporting your patients (whether in acute or in chronic disease) you should be especially careful to avoid throwing too much work on the digestive organs at any one time. Your supplies should be always administered in small quantities, more or less frequently repeated; never in a large amount at once. They should be well-timed, and the exact doses defined. When alcohol is being administered largely, animal food is best given in solution, as in broths or soups. The ability of the patient to take solid animal food may be regarded as the signal for diminishing the supplies of alcohol. Experience has taught me not to give two kinds of alcoholic fluid at the same time; do not give beer and wine, or wine and brandy; any one of them will agree better, because it will be more easily digested, when alone.

Patients often flush a good deal upon the first use of stimulants; this alarms the practitioner and deters him from prosecuting their further administration, or leads him to a vacillating practice, generally most injurious to the patient. It is a mere prejudice to suppose that any harm arises from this flushing of the face; generally it is an indication that the process of digestion, either of wine or spirit, or of other food,[1] is carried on with difficulty, and it will commonly cease by modifying the manner of its administration, such as giving less at a time, and more frequently. Sometimes, indeed, flushing will occur because an insufficient quantity is given, and an increase of the dose will get rid of it, just as an inadequate dose of opium disturbs the nervous system, whilst a larger one calms it.

In a word, I cannot too strongly impress upon you that, to do good with stimulants, you must use them early, with care and watchfulness, in very definite quantities, and not in a vacillating or timid manner. They are agents of inestimable value for saving life under all forms of acute disease, and I can say with truth, from a large experience, that the harm which they do (*in disease*) is grossly and unfairly exaggerated, and always due to the slovenly administration of them. The opponents of their use argue from their outrageous abuse in health, against their careful and scientific use in disease, forgetting how essentially different must be the effect of sixteen or twenty ounces of wine swallowed down within an hour or two, along with other food, and the same quantity carefully distributed in half-ounce and ounce doses over a period of twenty-four hours. I say it after mature reflection and a long course of observation, that there is no point of therapeutics so deserving of the study of the earnest-minded physician or surgeon, who is zealous to save life, as that of the action of these agents, both in health and disease.

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[1] The popular notion that alcoholic stimulants are not food, but a mere "flash in the pan," ought not to be encouraged by medical men in the present day.

# LECTURE IX.

## ON PYÆMIA.

GENTLEMEN,—The case upon which I propose to found my remarks to-day, is one which has afforded us great interest in our hospital visits for a considerable time; it is a good example of that remarkable form of disease which of late years has been called pyæmia—an affection remarkable in a pathological as well as in a physiological point of view.

CASE LXV. (Vol. xxxvi, p. 21.) The case to which I refer is that of a young man in Fisk ward, of the name of Gordon, who entered the hospital on the 27th of March, 1852. He is only twenty-three years of age, and of a strumous habit. Suppurative tendencies appear to have existed in members of his family: his father died from an abscess in the thigh, and his brother, some time ago, suffered from suppuration of the lymphatic glands on both sides of the neck; his mother died of phthisis.

The beginning of our patient's misfortunes was a gonorrhœa, which was followed, soon after, by a swelling in the perinæum; this increased, and became exceedingly painful; an abscess at last formed, which was opened in a Metropolitan Hospital, and about four ounces of pus escaped. The wound, however, did not close, but continued to discharge for a month afterwards. The gonorrhœa, which, up to this time, had not been attended to, was now treated and soon relieved; the abscess also closed, and appeared to be quite cured; the subsequent history, however, of the case renders it exceedingly probable that the suppurative process did not entirely cease, although the external wound healed perfectly.

On the 21st of March, he went down to Deptford, and indulged in beer and spirits more freely than he was wont to do. This intemperance was followed by fresh inflammation in the site of the abscess, with severe pain and swelling, and a sense of constriction around the anus. These symptoms were accompanied by fever, prostration, sweating, and pain in the joints, so severe as to prevent the patient from moving his limbs; the region of the abscess swelled considerably, and the pain and tension of the soft parts became very great. At this time, he was seen by a medical man, who considered that he was laboring under an attack of rheumatic fever. In a day or two, he was removed to the hospital, and his condition was as follows: He had a rapid pulse, and was sweating profusely; the tongue was foul, and there was an aphthous condition of the throat; the patient was very weak, and fast sinking into a low typhoid state; an abscess had formed in the perinæum, and his strength was rapidly failing. Such a train of symptoms indicated the necessity of support, and we therefore at once put him upon a liberal system, and supplied him with nutritious food and stimulants.

Although the constitutional derangement resembled very much that of rheumatic fever, we were not long in determining that the patient was not suffering from that affection. The history of the access of his illness was not that of rheumatic fever, neither was there any indication of the presence of the rheumatic diathesis. The existence of the gonorrhœa and the previous inflammation, although they did not negative the idea of the rheumatic nature of the complaint, at least pointed to another more probable explanation of his symptoms.

On April the 6th, symptoms of pleuro-pneumonia manifested themselves: a friction sound was heard at the base of the left lung behind; in fact, we had here an excellent example of a dry rubbing sound, audible both upon inspiration and expiration; at the same time we found crepitation at the base of the right lung. The nature of the case became cleared up by what took place on April the 10th, when the symptom manifested itself, which could leave but little doubt of the true origin of the other symptoms from which our patient suffered. At the outer border of the right ulna we found a collection of fluid in the areolar tissue,—in fact, there was an abscess of considerable size, in that situation. Upon further examination, we discovered two similar collections of fluid, in the subcutaneous cellular tissue, over the lower angles of both scapulæ. All these abscesses were immediately opened, and a free discharge took place from the wounds.

On the 13th, the lung symptoms increased in severity: the respirations had risen to 30, and the friction sound at the base of the left lung was very distinct; crepitation was still audible at the base of that lung,—it had become soft and loose in character, and almost amounted to gurgling; the breathing in the same situation was tubular, and the percussion dull. The other symptoms continued much as before: the hectic fever did not abate, there was still profuse sweating, and the pulse remained quick. At the same time, the patient's strength appeared to be gradually improving, which favorable alteration was no doubt due to the brandy and nutritious broths, which were administered as freely as his stomach would bear them.

Towards the end of April, free expectoration commenced, consisting chiefly of a considerable quantity of pus. On May the 12th, an abscess formed over the left hip, which was opened, and still continues to discharge. On the 5th of June (I pass over the daily reports of the case, thinking it better to mention the most important circumstances alone), a swelling appeared in the left buttock—an indication, no doubt, of the formation of another abscess, either in the gluteus muscle itself, or in the cellular tissue between two of the muscles. The occurrence of this swelling was accompanied by increased fever and debility. Although the abscess on the hip continued to discharge freely, the lung symptoms had much improved: the breathing had diminished in frequency, the crepitation was less, and the friction sound had almost disappeared.

The question now remains to be answered, whether further abscesses will yet form, and whether the patient's strength will be sufficient to carry him on towards convalescence. Our best chance of success will be by supporting his vital power with plenty of nourishment, in which I include alcoholic food.

Now, what is the rationale of this case—this febrile condition and this low typhoid state, accompanied by the formation of abscesses in different parts of the body? It belongs to a class of cases, which, though originating under various circumstances, and differing in the severity of their general symptoms, in the extent and situation of the local phenomena which usually characterize them, and in their duration and ultimate termination, are nevertheless alike in their essential nature. This consists in a poisoning of the blood by an admixture with it of purulent matter—either pus entire, and in its purest form, taken up directly into the circulation by an open vessel, or generated in some part of the vascular system; or unhealthy pus—decomposing, acrid, septic, received in a similar manner; or some of the amorphous elements of such pus, received by reabsorption into the vessels; or, some other animal matter, allied perhaps to pus, the product of disease during life, or of decomposition after death; or, lastly, a peculiar animal poison derived from the recently dead human body, or that of some other animal.

All these contaminations of the blood seem capable of giving rise to nearly the same train of general and local phenomena, similar to those from which our patient Gordon has been suffering. In the most severe cases, the disease begins with rigors, which are followed by fever of a low type, with utter prostration of strength, and the very rapid production, in quick succession, of circumscribed purulent formations, or *deposits*, in connection with various capillary systems, followed by increasing exhaustion, sweats, collapse, and death. In less severe cases, such as that of our patient Gordon, the course is slower, but the symptoms are similar: the fever is of a typhoid character, while the local suppurative processes tend continually to increase the exhaustion. To all such cases the name pyæmia—literally *pus-blood*, more freely *pus-contaminated blood*—is applicable.

It does not appear necessary, however, for the production of many of these symptoms, that the matter introduced should be pus, or even very closely allied to it: it need not be organized or even organic; inorganic substances, introduced into the blood, have produced phenomena very similar to those of the lesser grades of pyæmia, as has been proved by a variety of experiments.

The severity of a case of pyæmia depends, perhaps, in part, on the amount of morbid matter taken up, but probably much more on its quality—organic matter from dead bodies originates some of the worst cases of this kind—hence, as dissectors, you have a direct interest in this disease. The severity is also partly dependent on the previous general condition, or diathesis, of the patient; otherwise it would be difficult to assign a reason, why in one person, the reception of pus into a vessel, gives rise to the formation of a permanently limiting coagulum, while in another this coagulum is not formed, or rapidly breaks down into a pus-like fluid, and diffuse purulent infection results.

Among the most important experiments made on the lower animals, bearing upon the pathology of this disease, are those of Gaspard and Cruveilhier, who injected various substances into the blood of dogs, such as different kinds of animal matter in a putrescent state, pus, insoluble powders, &c.; these were introduced into a vein, and being carried into the circulation, created obstructions in various parts of the capillary system. One of these experiments of Cruveilhier is well known: it consisted in injecting ink into the femoral vein of a dog. The limbs soon became œdematous, indicating some obstruction to the venous circulation, the obstruction no doubt being caused by the coagulation of the blood in the large vein, in consequence of which the capillary system, which supplied this vein, became obstructed, and the watery part of the blood transuded through the coats of the capillaries into the cellular tissue, producing the œdematous condition. At the same time, the areolar tissue and muscles became the seats of little extravasations of blood, or, as they were described by Cruveilhier, little apoplectic clots; and these extravasations became the foci of purulent formations. A similar course of events has occurred in our patient, as in the subject of Cruveilhier's experiment.

Since the experimental introduction of pus, and other foreign materials, into the blood, gives rise to a peculiar train of symptoms, nothing can be more reasonable than to assign a similar train of phenomena, in cases where there is a manifest source of pus, from which the blood might be contaminated, to purulent infection; and even in other cases, where there is no *obvious* source of pus, and no other mode of explaining the symptoms, we may attribute them to a like contamination of the blood by morbid matter, received from some hidden local source, or generated within the vascular system.

Let us consider, rather more fully, the different sources of infection in cases of pyæmia, and the nature of the secondary pus-formations.

That form of phlebitis, generally the result of some injury to a vein, accidental or surgical, in which there is a tendency to suppurative, rather than adhesive inflammation of the vessels is the most direct and the most frequent cause of pyæmia. This was ably demonstrated more than twenty years ago by Mr. Arnott, a former Professor of Surgery in King's College, in an essay on the secondary effects of inflammation of the veins, which I strongly recommend you to peruse. You will find it in the fifteenth volume of the "Medico-Chirurgical Transactions." The recent experiments of one of the surgeons at this hospital, Mr. Henry Lee, and others, have shown that pus, and other foreign matters, introduced into the blood, often induce coagulation of that fluid around them, leading to the obstruction of the vessels, and subsequent limitation of the purulent infection to a very small portion of the vascular system, and its effects to the immediate locality. It is thus, in fact, that nature operates most effectually for the prevention of a diffuse contamination of the blood, and reduces the severe cases of pyæmia to comparatively few, notwithstanding the frequent coexistence of injured or open veins, and suppuration in or about them, in surgical cases. Should the state of the blood, however, be such that its coagulation by the pus is very imperfect, or should the coagulum, as sometimes happens, pass rapidly into a state of fusion, breaking down into a fluid having many of the characters of pus, there is no longer anything to prevent the free circulation of the morbid material, and its rapid admixture with the blood mass.

There are certain surgical cases which seem more particularly prone to give rise to purulent infection. These are, injuries and operations on bone, and the collection of pus pent up, and, it may be, decomposing, in sinuses and abscesses, especially in localities where veins abound.

In the former cases there is danger of pyæmia apparently for anatomical reasons. The veins in bones are patulous, their mouths being kept open by the adhesion of the walls of the vessels to their bony channels; moreover, it appears from the experiments of Cruveilhier, who injected the cancellous tissue of bone with mercury, that fluids introduced into the cancelli find a ready passage into the osseous vascular system; hence pus and other matters may gain admission to the circulation by the same route. In the latter cases, the particular diathesis of the patient will often favor purulent infection, by interfering with the natural occlusion of the vessels, and subsequent limitation of the morbid agent, as described by Mr. Lee. To this class the case of Gordon belongs. The abscess in the perinæum doubtless furnished the pus. In that situation there are numerous veins communicating both with the system of the portal vein, and also, through branches of the internal iliac, with the inferior cava and general circulation. It is doubtless owing to this disposition of the venous system that we so often find pyæmia following the perinæal section of stricture, and that abscess in the perinæum is so often the source of purulent infection.

But perhaps the puerperal state is, above all others, that most favorable to the production of pyæmia, or an allied disease. After the womb has expelled its contents, there remain on its surface many open mouths of large veins, only plugged by coagula, and most favorably situated for the absorption of any morbid matter which may be present, pus, decomposing discharges, or putrescent remains of the ovum. Or the mouths of the veins themselves may become the seat of inflammation and the formation of pus, which, under certain circumstances, may find a ready entrance into the circulation.

Cases of purulent infection sometimes originate from erysipelas, when the inflammation has terminated in suppuration, and collections of pus have formed in the skin and areolar tissue.

Lastly, one of the worst forms of pyæmia is apt to arise in cases of typhoid fever, from the absorption, I believe, of some of the products of the sloughing and ulceration going on in the bowels. A case of fever may be doing apparently very well, when suddenly the typhoid symptoms become greatly aggravated, and the patient rapidly sinks.

Lastly, you must class with these cases the formidable disease which follows wounds received in dissection, which formerly used to prove so fatal, and was the cause of the loss to science of many men of high promise, who were cut off by it at an early period of life. Wounds poisoned by diseased fluids, received from the living, are likely to produce the same train of symptoms, and to lead in the same way to a fatal result. (Case LXVIII.) I need not say how much you are each and all of you personally interested in the disease as originating in such a source.

The mode of formation of secondary pus-formations, or *purulent deposits*, or *metastases*, as they have been called, has given rise to considerable differences of opinion among pathologists.

These pus-formations are found in all parts of the body, and the development of them in great numbers, with sometimes extraordinary rapidity, in various parts successively, forms one of the most remarkable features of the higher grades of pyæmia.

First, they are found in the parenchymatous organs, especially the lungs and liver, where they appear to commence, in the first instance, as red capillary obstructions, with solidification of the textures around, and followed immediately by sloughing or suppurative fusion of the whole patch involved; so that a collection of pus is the result, with ordinary inflammation immediately around it.

Secondly, effusions of pus may take place on the surface of serous membranes, as the peritoneum; but they are far more frequently met with in the synovial sacs, and they there lead to the corrosive disorganization of the joints. Sometimes we find a rapid development of pus on the surface of mucous membranes, the bronchial for example, giving rise, in that case, to a copious purulent expectoration.

Lastly, and very commonly, the muscles and areolar tissue are the seat of secondary abscesses.

What determines the particular situation in which these pus-formations are found, in any individual case, it is not easy to conceive; but generally it seems that the first capillary system through which the infected blood circulates, becomes the seat of the formation of abscesses. In most instances, the first system is that of the lungs, and here we shall generally find evidence of capillary obstructions. This has been found, both in the experiments intentionally performed on animals, and in those which we unfortunately have many opportunities of seeing performed by disease on the human subject. In other cases, where the portal venous system is primarily infected, the secondary abscesses form first in the liver.

Some pathologists have considered these pus-formations to originate in the mechanical obstruction of the capillaries by aggregated pus cells, or by small masses of coagulated fibrin, called *emboli*, too large to traverse them, and the subsequent mere deposition of pus there, or its multiplication from the blood elements. But this view, so purely mechanical, does not, to my mind, fully account for the rapid breaking down of the textures around the obstruction by an active inflammatory process. It is not necessary that pus should be introduced into the blood to give rise to these pus-formations: other fluids injected into the blood, as shown by the experiments of M. Gaspard, have produced them. And, therefore, it seems to me, we must look for some process of *contamination* of the tissues surrounding the obstructed point, to account for their destruction with inflammatory phenomena, and to explain the attendant constitutional disturbance. The obstruction by *embolus*, or in any other way, explains the localization; the depravation of nutrition by some material of the nature of a poison, is needed to explain the inflammatory process in the surrounding texture; unless, indeed, that process be considered as the necessary result of the death of a certain portion of tissue from the sudden or rapid stoppage of its blood supply.

After all, it seems to me quite as difficult to account for the very rapid formation of the little circumscribed slough, which constitutes a common boil, as these purulent deposits.

When we consider, that although the morbid matter introduced into the blood, has failed to cause coagulation in the vessel through which it gained admission to the circulation, it may, nevertheless, succeed in obstructing the capillaries in that way, and becoming fixed there, it may act as a local irritant; and when we consider also the terrible effect produced on the whole system by the contaminated blood, as though by some deadly poison, depressing the vital powers of the patient to such an extent as sometimes to cause death, independently of, and prior to, any secondary local effects, it need not be a matter of much surprise, I think, that the local irritation should determine a very asthenic inflammation, passing rapidly into sloughing and the formation of pus.

I have one or two more remarks to make on Gordon's case. You will recollect that, on April the 13th, we had dulness over a portion of the chest: in the situation of the dulness we heard coarse crepitation, and, after a time, crepitation almost amounting to gurgling. The matters expectorated contained a large quantity of pus, and I thought that purulent infiltration had taken place, and that the formation of a cavity was imminent; indeed, there can be no doubt that a rapid formation of pus did take place, as in pneumonia, and that it was as quickly eliminated by expectoration, but without destruction of lung-substance.

We had other indications of the occurrence of inflammation within the cavity of the chest, in the presence of a rubbing sound on the right, and also on the left side, showing that there was a roughened, or at least a dry state of the pleural membrane in these situations. Here, then, we had both the systemic and the pulmonary capillaries affected with inflammations, which afterwards became the seat of collections of pus. The case exhibits, in a most striking manner, phenomena precisely similar to those which are met with in the subjects of experiments, when pus is injected into the veins of living animals.

For practical purposes, we may, I think, divide these cases of purulent infection into three classes:

The first class of cases is distinguished by this common character, namely, that the course of the disease is very rapid and the result is certainly fatal. As an illustration of this class of cases, I may adduce the experiment of Gaspard, which consisted in injecting into the jugular vein of a living dog three drachms of healthy pus. In three minutes afterwards the dog was seized with copious vomiting and micturition. For a quarter of an hour the limbs were violently convulsed, and complete emprosthotonos frequently occurred. The subject of the experiment died five hours after the pus had been injected into his vein, in a state of extreme exhaustion. Now, in such a case, the fatal result can hardly be explained by the supposition that a great number of local obstructions have taken place, for there can hardly be time for such an occurrence, and it seems more reasonable to attribute it to a poisonous influence acting on the nervous system in a manner somewhat similar to that by which we explain death from prussic acid, and other violently poisonous substances. In such cases, it appears that death results from a general contamination of the whole system by the noxious matter, rather than from the occurrence of local mischief.

In practice we have such cases—cases in which death results in two or three days, or even less, from the first circulation of pus in the blood. Let me give you an instance:—

Case LXVI. (Vol. ii, p. 135.) A young woman named Mary Riley, twenty-one years of age, was admitted into the hospital under my care, January 25th, 1841, in consequence of a black gangrenous appearance and ulceration of the lips, especially the lower one; there was also much swelling and redness about the left eye.

She stated that the soreness of the lip commenced about a week before, with a small pimple, which gradually increased, and was followed by great swelling, and a black-looking ulcer at the left angle of the mouth. She had been, she said, in good health previously.

The day after her admission, the pulse became exceedingly rapid and feeble; she complained of great thirst and much pain in the lip. Rapid collapse ensued; there was no time for treatment, and she died the following night.

On examining the body after death, I found the cellular tissue of the lip much infiltrated with pus; much hardness and swelling about the angle of the jaw on the left side, involving the salivary glands; incipient inflammation of the left jugular vein; the blood imperfectly coagulated and adhering to the lining membrane; the coagulum of a dirty brown color; pus in the smaller veins. The uterus and ovaries were highly vascular, the latter much enlarged; there were several very large Graafian vesicles, with red inner membranes, one containing a small vesicle (germinal ?) surrounded by granular matter.

These fatal cases are unfortunately too frequently met with in the puerperal state, and often come on very insidiously. A woman goes through her confinement in the most satisfactory manner, and all appears to be going on well, when formidable symptoms of rapid prostration manifest themselves, and the patient dies in one or two days. In such a case the pus enters the circulation probably by the medium of the uterine veins, and fatal exhaustion, frequently accompanied by convulsions, is soon induced, as in the experiment of Gaspard. Lately I saw a lady whose confinement, I was told, had been perfectly natural, and a month had passed over in a satisfactory manner; in fact, she had been out for a short time, and everything seemed progressing favorably; but on the evening of the day after she went out, she was seized with a severe rigor, which was followed by fever, and rapid prostration, and in eight-and-forty hours she was dead. I was present at the post-mortem: we could find no evidence of peritonitis, nor any signs of active inflammation in the cavity of the uterus, but there was evidence of phlebitis affecting the large uterine sinuses.

There are other cases of this kind, closely allied to the last, in which a rapidly fatal result occurs: these are cases of puerperal fever.[1] All fevers, I hold, are due to an alteration of the blood, induced by the introduction into that fluid of some morbid matter. It is not often that we have an opportunity, in this hospital, of observing puerperal cases from their commencement. The poor woman, the subject of the following case, was confined in the hospital, and the notes taken comprise the whole history.

Case LXVII. (Vol. xxi, p. 30.) Hannah Donovan, a married woman, 19 years of age, obtained admission to the hospital in consideration of her having a tumor in the abdomen, which we soon discovered to be a gravid uterus, and shortly afterwards our diagnosis was confirmed, by her being delivered of a healthy child, July 18th, 1847.

Everything went on satisfactory until the 26th, when she was attacked with symptoms of fever; great thirst and languor, a dry and brown tongue, and a rapid and feeble pulse. At the same time the secretion of milk diminished, and there was considerable abdominal tenderness, while her whole appearance was remarked as characteristic of the terrible malady from which she was suffering, namely—puerperal fever. Turpentine stupes were applied over the abdomen. By the 30th, though there was less abdominal tenderness, she had become much worse: her face wore an expression of great anxiety; there was great heat of skin, a parched mouth covered with sordes, and a furred tongue; she was only partially conscious, and sometimes delirious; pulse 129, and very feeble, respirations 30; a mucous rattle was heard in her throat. Some eight ounces of wine were given in the day, and carbonate of ammonia; but she continued to get worse, and died on the night of the 31st. The following is the record of the post-mortem:—

"The uterus was found as large as a man's open hand, flattened on the left side, from the pressure of an enlarged ovary.

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[1] Dr. Robert Ferguson, formerly Professor of Midwifery in King's College, was the first, or among the first, to put forward, distinctly, what is now generally regarded as the true pathology of puerperal fever—namely, blood contamination from vitiated uterine secretions. See his work on Diseases of Females, published in 1839. I have also discussed the subject in my Croonian Lectures on Gout and Rheumatic Fever, delivered at the College of Physicians in 1843.

On section, pus was found to exude from the veins; the walls appeared semi-cartilaginous, and at the upper and back part of the cavity, a large, irregular, suppurating surface appeared, about the size of a crown piece; another similar, but smaller one, surrounded the opening of the left Fallopian tube, and several, yet smaller, were found on the inner surface of the enlarged ovary. The peritoneum covering all the pelvic viscera was much congested throughout, presenting appearances of incipient inflammation. The kidneys rather large and congested; the ureters much dilated—half an inch in diameter—but equally through their whole length, and presenting no pouch." There was some evidence of pleurisy on both sides. A large fibrinous clot occupied the right ventricle.

I shall here introduce another example of pyæmia which proved quickly fatal. In this case, pus was found in some of the veins, but its source was by no means obvious. The lungs were in a state of patchy hepatization: these patches may have been the earlier stages of secondary pus formations.

Case LXVIII. (Vol. xiii, p. 160.) Elizabeth Miller, æt. 42, had been assisting one of the nurses in the surgical wards, and was attacked, without any assignable cause, with shivering, headache, utter distaste for food, and vomiting. These symptoms were followed, a day or two after, May 15th, 1844, by great pains in the joints and bones of the face, with entire prostration of strength, a rapid pulse, 112, and quick oppressed breathing, 34. Both wrists then became very red and tender, and there was a slight blush on the left leg. On the 17th, the left leg and several joints remained acutely painful; sleeplessness, and the same general symptoms, continued. By the 19th, she was much weaker, and despite an ounce of brandy which was then ordered every hour, the prostration increased, the pulse became more rapid, 132, the surface bedewed with perspiration, while her body remained everywhere warm; and though she rallied temporarily under the stimulants, she rapidly sank again, and died the same night.

At the post-mortem examination of the case, the appearances found were:—Some comparatively recent pleural adhesions, but no marks of recent inflammation. When the lungs, which were somewhat congested, were cut into, several small circumscribed patches in a state of red hepatization were found. There were two inactive hydatid cysts in the liver. When the right renal vein was cut into, a quantity of pus, tinged with blood, made its escape.

Sometimes we have opportunities of observing cases in which the introduction of pus into the circulation is as certain and direct as when injected into the veins in experiments. In a former lecture (page 83) I described two cases of this kind, in one of which the source of purulent infection proved to be an abscess at the septum of the heart; in the other, an ulcer at the base of one of the mitral valves.

Another class of cases, in which the result is often rapidly fatal, comprises those which arise in the course of erysipelas, particularly idiopathic erysipelas of the head and face, which more especially concerns us as physicians. A case may be going on in a manner apparently quite satisfactory; the pulse may go down, the tongue become clean, and the prospect of recovery may be looked upon as certain, when suddenly rapid prostration comes on, the patient becomes comatose, and often within twenty-four hours is dead. Now, in such a case, it is difficult to prove that the fatal result depends upon the introduction of pus into the blood, but we know that in erysipelas there exists a great tendency to the formation of pus; and when we consider the great similarity of such a case to those in which we have decided evidence of the introduction of pus into the circulation, it seems not unreasonable to refer the fatal result to the same cause.

Let me give you an instance of pyæmia following traumatic erysipelas of the head; the superficial closure of the wound probably preventing the free escape of matter. The case illustrates some other points in the pathology of pyæmia: there were numerous pus-formations in the lungs, with the circumscribing inflammatory areola well developed. Profuse expectoration of pus was the chief feature of the case, which, I have little doubt, was derived chiefly from the bronchial mucous surface.

Case LXIX. (Vol. xvii, p. 37.) Patrick Shannon, an Irish laborer, was admitted into the accident ward of King's College Hospital, with symptoms of concussion and a scalp wound, the consequence of a severe blow on the head. He recovered from the immediate effects of the blow, but erysipelas attacked the wound, and spread to the head and face. From this he seems to have recovered only partially: the scalp wound healed deceptively and on the surface only, and he then began to cough and expectorate a large amount of purulent matter, for which he was removed to the medical wards, and placed under my care, May 2d, 1846. He was then much emaciated, and his breath and whole body exhaled a sour and sickly odor of pus. The pulse was 136; the respiration short and quick—about 36; he had a constant hacking cough, with expectoration of true pus. On percussing the chest, it was found generally somewhat dull. There was one patch of much more marked dulness behind, about the angle of the right scapula; and at the same spot crepitation was heard of a mixed character, with gurgling on coughing. Elsewhere, varying crepitation and rhonchus, with breathing, sometimes puerile, sometimes tubular, were heard. Carbonate of ammonia and brandy were ordered.

On the next day, the 3d, the same symptoms continued in an aggravated form: the same copious expectoration of pure pus; precisely similar, but more developed chest sounds; the same rapid pulse and breathing, numbering 132 and 42 respectively; some delirium. On the 4th, the symptoms were nearly the same, but of a still more typhoid type; there was great heat of surface, and some reddish discoloration of the integuments about the chest had taken place—a not unfrequent occurrence in these cases. The pulse and breathing were 160 and 52 respectively. The chest signs were similar to those on the 3d, but more developed. I shall not trouble you with the long detail of them. The bowels showed some tendency to become relaxed, which was greater the following day, and several watery evacuations were passed. On the 6th, there was increased delirium; the same copious expectoration of pus continued, and his breath was very offensive; the other symptoms were much the same as before. The brandy was now increased to three drachms every hour. On the 7th, he was much worse: more exhausted and feeble; and he died that evening.

The error in the treatment here, consisted in putting off the free administration of brandy and nutriment until the eleventh hour; though it is, I think, highly improbable that this patient would have recovered under any plan of treatment, for the disease was of a high grade, and the local disorganization which it gave rise to was very great, as the following results of the post-mortem examination show:—

On opening the chest, the lungs *appeared* natural, though perhaps slightly emphysematous; but on cutting into them, numberless small cavities were found in clusters, filled with pus, and surrounded by lung tissue in a state of red hepatization; the bronchial mucous membrane, towards the base of each lung, was red, soft, and velvety; there were some pleuritic adhesions. The brain and the other viscera were healthy.

Another set of cases of this kind are those in which this train of symptoms follows severe injuries or operations, more especially operations on the bones, as I have before mentioned.

A very unfavorable termination not unfrequently occurs after the operation of lithotomy, and after division of old strictures, where the case has been progressing favorably up to a certain period (the fever becoming less and the wound looking healthy), when fatal prostration comes on, and it can hardly be doubted that the sudden change is due to the introduction of pus into the circulation, the symptoms agree so closely with those of cases of pyæmia.

The same train of symptoms is sometimes brought about by the absorption of pus from Peyer's glands in a suppurating state.[1]

In a second class, I would include those cases, in which the result is no less fatal, but in which the course of the malady is extended over a much longer period of time. Many of these cases are also puerperal, and have been described by some French authors as rheumatic in their origin. These cases commence with tenderness over the region of the uterus, followed by thirst, heat of skin, fever, and swelling of one or more of the joints. The sterno-clavicular joint is that which is, perhaps, most frequently first affected, then the shoulder, and soon afterwards a knee-joint becomes swollen and tender. When you find the sterno-clavicular, or any other joint very full of fluid in a puerperal woman, you must not at once set it down as dependent upon a rheumatic condition, but must anxiously watch the case, and hesitate to pronounce a favorable prognosis, lest it should be followed by the formidable symptoms I have described.

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[1] For the further illustration of this form of pyæmia, see the case of John Gavin, Case XVI, p. 99, in the present volume; and the remarks upon it.

You may sometimes see every joint in a patient's body thus filled with purulent fluid. The pus circulating in the blood probably creates obstructions in the capillaries of the joints, which afterwards become foci of pus-formations, as we have seen in other cases. The strength of the patient soon fails, and in spite of your best-directed efforts, you are unable to prevent a fatal result. On examination, you find many of the joints quite full of pus, and the synovial membrane, and frequently the articular cartilage destroyed. Sometimes the ends of the bones are laid quite bare, and the cartilage dissolved in the purulent matter: occasionally also pus is present in the muscles. The result is uniformly fatal, although the affection may run a course of several weeks.

The annexed case is a good illustration of what I have just said. Here the joints were the seat of the secondary pus-formations, and disorganization was rapid and complete. The symptoms at first were mistaken for those of rheumatism, and this, I think, is not an uncommon error of diagnosis, which indeed had been at first committed in the case of our patient Gordon.

Case LXX. (Vol. ii, p. 186.) Ann Davies, a married woman, 28 years of age, after a severe and protracted labor, and much neglect and bad nursing, suffered from what was, at first, supposed to be an attack of rheumatism. Her medical attendant (Mr. Dunn) suspected that "the veins on the left side were also affected." She was admitted into Augusta ward, February 24th, 1841, about six weeks after her confinement, with the following symptoms:—

"Great dyspnœa; a pale and anxious countenance; considerable anasarca of the lower extremities; a small and rapid pulse. There was a tumor, about the size of an orange, over the right sterno-clavicular joint, soft and fluctuating, and evidently containing pus. On moving the arm, a grating sensation was felt in the shoulder-joint of that side."

Stimulants were administered, but with no benefit; she became very delirious during the night, the dyspnœa more urgent, and she died the following morning.

After death the abscess was found to involve the sterno-clavicular joint, the cartilages of which were entirely destroyed, and the end of the clavicle was softened. The abscess extended among the muscles. The shoulder-joint was found in a condition similar to that of the sterno-clavicular, and several ounces of pus escaped. The left knee-joint also contained a small quantity of sero-purulent fluid, and a false membrane. Pus was also found in the left sacro-iliac joint. No purulent deposits were found in the lungs or other parenchymatous organs, but the bronchial tubes contained some sero-purulent fluid. Neither the uterus, peritoneum, nor veins generally, presented any morbid appearances. The heart contained firm coagula in all its cavities, adhering pretty closely to the cardiac walls.

The third class of cases is much less fatal, and to this belongs the case of our patient Gordon. The less frequently fatal termination of cases of this class appears to be due to the different parts of the capillary system affected. In our patient the deposits have chiefly occurred in the cellular tissue and muscles; and the result will probably depend upon the state of the lungs and the patient's strength. I believe that the lungs are fast recovering their healthy condition, and therefore he has only to contend against the external formation of pus. If we can keep up his strength long enough for the large abscesses on the hip and buttock to fill up and close, and if no fresh collections of matter occur in other parts of the body, we may confidently look forward to a favorable termination.[1]

I will now add an instance of the successful treatment of a somewhat different example of this class:—

CASE LXXI.[2] (Vol. lviii, p. 15.) Sarah Butcher, a nurse in our hospital, after having been in attendance on some very bad surgical cases, became generally ill, and suffered from pain and aching in her limbs, and slight sore throat; at the same time her right thumb became inflamed, for which she could assign no cause, as she did not remember to have had any excoriation or wound. An incision was made, but no matter escaped. A small swelling then made its appearance in the axilla, but quickly disappeared again. At first she was ordered a mixture of ammonia and chloric ether three times a day, a little morphia at night, and poultices to the thumb. On the 30th of April, her pulse was 104, and her tongue covered with a thick blankety fur. Half an ounce of brandy was ordered every two hours, five grains of compound soap-pill at night, and, as her bowels were confined, a purge.

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[1] This man ultimately recovered; and in August was sufficiently well to leave for a convalescent institution.

[2] Reported by Mr. H. F. Winslow.

After this, the whole hand became swollen, tender, and extremely painful, so as to prevent her sleeping; and on the 3d of May, slight fluctuation was felt under the palmar fascia; her pulse was 112. On the following day she was seen by Mr. Fergusson, and by his advice the house physician applied ten leeches. Her pulse and breathing that day were 116 and 36. During the three following days her hand and arm continued very tender and painful, and much swollen. On the 5th, the pulse was 104; on the 6th, 102; on the 7th, 96. A peculiar rose-colored vesicular eruption became developed on the 7th over all her body; she complained of pain in her joints; the brandy was increased to twelve ounces; an incision was made in the hand, and though no pus escaped at the time, yet it seemed to afford some permanent relief from pain, and by the 11th, there was a free discharge of pus from the wound. She was now taking one grain of opium every four hours.

On the 13th, a very great rise in the pulse accompanied a fresh formation of matter in the arm, and declined after its evacuation by incision. The wounds continued freely discharging, but on the 17th the pulse again became more frequent, and on the 18th the brandy was increased to twenty-four ounces, *i. e.*, one ounce every hour. On the 22d, a fresh exit was given to the matter by an incision in the palm. There was still great pain.

On the 24th, the opium was ordered to be given in the form of tincture with bark. On that day the pulse was 92; on the 25th, it had risen to 100; on the 26th, to 108; on the 28th, to 118; on the 31st, to 124. There was no general change for the worse corresponding with this increasing pulse; but on an incision being made over the back of the wrist, and a quantity of matter let out, the pulse fell, in a few hours, from 124 to 84; her tongue became quickly clean, the pain greatly diminished, and she improved generally. Soon afterwards the discharge of matter ceased, and cold water dressing was substituted for the poultices, which had hitherto been constantly applied. The brandy was reduced, on the 2d of June, to eighteen ounces, and as the improvement continued, it was still further reduced to twelve ounces on the 4th, and to eight on the 6th, and then discontinued altogether, and some porter substituted. She suffered from wandering rheumatic pains for some days, but otherwise improved rapidly, and was discharged on the 28th of June, with of course, considerable impairment of the hand and arm.

This patient had altogether about 31 pints of brandy—or about an average of a pint a day, for a month.

The following case is a third example of the same class. It has many points of interest, and the diagnosis was difficult. The symptoms at first closely resembled those of acute rheumatism; but, looking back at the complete history, the true nature of the case is sufficiently obvious. I am sorry to say the treatment was not such as I should now recommend: it amounted, in fact, to almost nothing, excepting the local measures, and to this I attribute the very tedious character of the illness, and the slow convalescence.

Case LXXII. (Vol. xvi, p. 17.) Caroline Allen, æt. 32, on October 6th, 1845, was attacked, without any assignable cause, with shivering, fever, and oppression; and in the evening observed a red spot on the forefinger of the left hand, which produced much pain, and was followed by redness and swelling of the hand and arm. The red spot on the finger was lanced by a surgeon who supposed it to contain pus, but no matter escaped. The next day the right hand and wrist became painful and swollen, and then the left foot and ankle. All these joints were swollen and tense when she was admitted, on the 18th. Her pulse was then 120, and the respirations 28: the skin moist; the tongue furred in the middle; there were cough, and expectoration of serous fluid, with purulent pellets and some clots of blood, but no corresponding abnormal sounds were heard in the chest. The case at first was treated as one of rheumatism, a blister applied to one wrist, and Dover's powder given.

On the 12th there was increased fever; the right arm was much swollen and covered with a blush of erysipelas; as also the left leg, but in less degree. Warm fomentations were applied, and a little morphia given at night. The next day, as the erysipelatous redness continued, the left ankle and foot were scarified.

From the 14th to the 18th, the same symptoms continued with little alteration: pain, superficial redness, swelling, and œdema, varying in intensity, about the left foot and ankle, and the right wrist and hand; and a pulse of 112. On the 18th, the pain in the left foot and ankle was very severe, and the palm of the right hand became swollen, prominent, and red; the swelling extended along the palmar surface of the fingers, but no distinct fluctuation could be detected in the palm. The same expectoration of mucus and pus with small clots of blood continued, and a pulse at 120. A grain and a half of quina were now ordered three times a day.

On the 20th, the limbs were placed on splints; at first without, then with bandages; cold lotions were applied, and some wine and good nutritious food were ordered about this time. Still the same parts remained very painful and swollen; the pain at times was much aggravated, and at others abated; the pulse continued weak and rapid, varying from 108 to 120, and very little progress was made for many weeks. On the 8th of November, an issue was established over the right wrist, and some time later a second. Now and then we could feel, more or less distinctly, fluctuation about the joints, especially the wrists; the cough and expectoration continued without corresponding physical signs in the chest.

About the 21st of November, our patient began decidedly to amend: the pulse improved and diminished in frequency, and the local symptoms subsided. She continued on a tonic plan of treatment, very slowly improving, till December 31st, when she was discharged cured. At first the joints, as one might have expected, were somewhat stiff and useless.

Before we part, let me say a few words upon the treatment of these cases. This may be summed up in one word—*support*. In fact, in these cases, there is a struggle between the depressing influence of the pyogenic process and the strength of the patient; hence, the more support we are able to give him, the more favorable will his position be to meet the struggle. You have seen this plan of treatment fully carried out in the cases of Gordon and Sarah Butcher. We also get a hint as regards the management of our puerperal cases, and we are practically cautioned against the fashion (now happily becoming extinct) of giving depressing medicines to puerperal women. The woman's vital powers after labor are more or less weakened; she has gone through what may be compared to a severe surgical operation, and to this the analogy of labor becomes more striking, when we consider that the part of the uterus to which the placenta was attached, resembles an extensive raw surface, or immense wound; the vessels are torn, and more or less hemorrhage must have occurred.

I believe that now-a-days experience decides in favor of upholding the strength in a moderate way after parturition; at least this seems to be the opinion of many accoucheurs to whom I have had an opportunity of speaking on the subject. The same remarks will apply to the treatment of severe surgical operations: I should imagine that it would be now very difficult to find a surgeon who would advocate the old plan of preparing a patient for an operation by bleeding, strong purging, and other very lowering treatment, as was formerly the custom. It is the same as regards the treatment of severe injuries, including burns; we must supply the patient with moderate support, and give him as much nutritious food as his powers of digestion will bear. In cases of erysipelas, we must always bear in mind the very doubtful nature of the case, and must avoid giving unhesitatingly, a very favorable prognosis, lest, when we least expect it, a formation of pus should occur, and some of the purulent matter entering the circulation, rapid exhaustion should follow, and our patient die under the depressing influence of the poison, just as takes place with the subjects of experiments.

# LECTURE X.

## ON PNEUMONIA AND ITS COMPLICATIONS.

GENTLEMEN,—We had lately had some cases of pneumonia treated with a highly favorable result, upon a plan which differs materially from that laid down by some of our highest authorities in the practice of medicine. I propose, then, in my present lecture, to make these cases the basis of some remarks on the treatment of pneumonia, with a view to solving the problem, what is the best mode of treating this disease; that is, what mode of treatment is best calculated to lead to a speedy resolution of the inflammation, with least injury to the patient's constitution, and with the shortest convalescence.

I will observe, *in limine*, that the plan of treatment which I have pursued in the cases now convalescent, as well as in many others, consists, not in the use of remedies directly antiphlogistic (so called), that is, of remedies intended directly to knock down inflammation by withdrawing blood, the supposed fuel of all inflammation, and by reducing vital power; but in the employment of means which will promote the free exercise of certain excretory functions, by which the blood may be purified, and certain matters removed from the system, which, remaining in it, tend to keep up a state very favorable to inflammatory affections. The remedies to which I refer, tend to promote the free action of the skin and of the kidneys, and, in a less degree, that of the intestinal mucous membrane; whilst, at the same time, a free stimulation is maintained of that part of the skin which is near the seat of the pulmonary inflammation; and an essential part of this treatment is, that while these remedies are being used, we do not aim at reducing the general powers of the system, but rather at upholding them by such frequent supplies of nourishment, easy of assimilation, as may be readily appropriated, and duly apportioned, both in quality and quantity, to supply the waste which during the inflammatory process must necessarily take place in the most important tissues of the body, especially the muscular and nervous.

Some physicians have drawn a distinction between cases of pneumonia, which is useful with reference to treatment. There are, they say, two classes of cases of pneumonia:—the one sthenic, the other asthenic and typhoid; the former capable of bearing the most active antiphlogistic treatment, and for which, indeed, they say, that that treatment is absolutely necessary; the latter requiring a supporting, and even a stimulating plan, and for which an antiphlogistic one would be extremely hazardous and dangerous. Now, while I fully recognize and admit the practical value of such a distinction as this, I must remark, that it seems to me it ought to be expressed differently. I would say that in all cases pneumonia has, independently of this or that mode of treatment, a decided tendency to depress the general powers of life—in some more, in some less; that, with all, a very decided direct antiphlogistic treatment is hazardous,—with some extremely so,—and in none is it absolutely necessary; but, with others, there is no safety for the patient, unless the treatment from the beginning be of a decidedly supporting and stimulating nature.

You will note the distinction which I make between remedies *directly* and *indirectly* antiphlogistic. The former is a class of remedies whose supposed efficacy is founded upon a notion (an erroneous one, as I think), that certain acute and sthenic inflammations are attended with an undue exaltation of the vital forces, both local and general, and that these must be reduced before the inflammation will yield. I say, I think this view erroneous; for it seems to me quite plain, from the clinical history of the malady, that the local inflammation draws so largely upon the rest of the system, as to depress the general powers of life; else, whence the weakness, the exhaustion, the failure of appetite, the wasting, which take place in the course of the disease, even when favorable, independently of any particular line of treatment?

The remedies indirectly antiphlogistic are those by which it is proposed to promote and exalt some particular functions,—as sweating, or some other secretion, which tend to purify the blood, by eliminating noxious matters through their proper channels, and by such purification of the blood, to reduce or remove febrile symptoms.

CASE LXXIII. (Vol. xxxii, p. 88.) The first of the cases to which I shall refer to-day is that of Edward Mills, aged 28. He is a railway porter—a good specimen of a strong, athletic man, of active habits, and accustomed to hard work, and, at the same time, evidently one who has been well fed. In short, he is a person just adapted, by constitution and habit, to bear the so-called antiphlogistic treatment, if such were necessary.

On the 3d of January, 1851, he was seized with shivering, headache, shortness of breath, and cough, soon followed by the occurrence of a sharp pain in the left side, particularly upon taking a deep breath, and all this accompanied by loss of appetite and fever. The next day he began to expectorate a quantity of very viscid and rusty mucus, the breathing became more rapid, and the cough more frequent and troublesome; on the 5th he came into the hospital.

The character of the sputa at once attracted our notice; and those of you who came round with me will recollect, that I particularly directed your attention to their extreme viscidity, and showed you how they adhered to the vessel in which they were received; so much so, that I could turn the pot upside down without the least displacement of its contents. We likewise observed the peculiar rusty color of the sputa. The matter expectorated in such cases looks exactly as if it had been mixed with iron rust. The peculiar color is caused by the intimate admixture of a certain amount of the coloring matter of the blood, which may be seen in it through the microscope. The mixture is evidently very intimate: there is, in fact, in these cases, a hemorrhage, doubtless from the naked vessels of the pulmonary air-cells. But the appearance and color of the sputa differ very decidedly from those of the expectorated matter in ordinary cases of hæmoptysis. I think the difference is to be explained thus: In pneumonia, a copious secretion of mucus takes place from the membrane of the extreme bronchial tubes or bronchial passages; and blood, escaping from several minute vessels, becomes intimately intermixed with the mucus, and gives it its rusty color. The escape of blood and the secretion of mucus take place simultaneously, and in nearly constant proportions, and are dependent on the same cause, namely, that which irritates the lung. But in hæmoptysis the escape of blood is independent of any secretion of mucus, and often takes place without it, and the quantity of blood is always greatly in excess of that of mucus.

When present this viscid and rusty state of sputa is a pathognomonic sign of pneumonia. Indeed, the great viscidity alone, even without any red color, but with a yellowish, bilious hue, ought always to excite our apprehension, lest pneumonia may be commencing. Passive congestion of lung, such as we may meet with in low fevers, or in heart disease, will, however, sometimes give rise to sputa not unlike those of pneumonia, but to be distinguished from them by their being less viscid, and containing more blood, which is much less intimately mixed with the mucus.

Although the rusty and viscid character of the sputa will often enable us, with certainty, to diagnose the presence of inflammation of the substance of the lung, we must not conclude that the absence of this characteristic expectoration is a positive proof against the existence of pneumonia. Many cases of pneumonia pass through all their stages, and resolution takes place, without the occurrence of any expectoration, or with that of a very trifling amount of colorless mucus. In the cases of typhoid pneumonia, it is not uncommon to find that the patient does not expectorate, probably owing to his weak and sluggish state, his sensibilities being much blunted; or the expectoration has peculiar characters, being much less viscid, non-adherent, and its color much darker, exhibiting an appearance which has been likened to that of prune-juice.

We found that our patient exhibited certain well-marked constitutional symptoms. He had a flushed face, a hot and dry skin, and a tongue coated with a thick white fur. He also complained much of thirst, and of a troublesome cough, and he suffered from a shooting pain in the left side, below and a little in front of the scapula, and extending over the shoulder, which impeded his breathing, and was increased by deep inspiration. At the same time there was no very great increase in the quickness of pulse (it was 96), but the respirations were much more frequent than in health, being 36. The pulse was full and strong, and such as would have not only justified, but invited bleeding, if we had allowed ourselves to be influenced by that single symptom in adopting such a line of treatment.

A careful examination of the chest at once enabled us to determine the exact nature of the evil under which this patient was suffering. By percussion and auscultation we found a normal state of the right lung, the breathing being, however, rather more intense (puerile) than in health. On the left side, the percussion sound was quite natural in front, and good vesicular breathing was audible. Such, likewise, was the case all over the scapular region behind. But at the base of the left lung, all about the region to which he referred his pain, a decidedly dull sound was elicited by percussion.

Now this dulness of percussion may arise, as you know, either from an effusion of fluid between the pleural membranes, or from a thickened state of pleura, or from a condensation or such other alteration of the lung as prevents its free distension by air. When fluid is interposed between the pleuræ, the vibrations excited in the lung by the voice, are not propagated to the wall of the chest, unless in cases where some old bands of adhesion serve to connect the surface of the lung with the costal pleura.

In the present instance, the vibrations were sensible to the hand over the dull surface, and, therefore, we had to seek some other cause for the dulness, besides pleuritic effusion. There was no history of any former attack of pleurisy; therefore it was improbable that there could have been any thickened state of pleura. We found, however, a decided modification of the voice and breathing, which sufficiently accounted for the dull percussion. There were both bronchophony and bronchial breathing. All these conditions occur only in a solidified state of lung; and when accompanied by viscid, rusty sputa, and the train of symptoms which we found in our patient Mills, we have no hesitation in referring the solidification to the effusion of plastic matter into the air-cells of the lung, which likewise serves to exclude the air from them.

The seat of the effusion of plastic matter in pneumonia is in the fine air-passages within the lobules of the lung, the interlobular passages being free. The air, therefore, in inspiration rushes through these tubes; and the vibrations which it excites on the walls of the tubes are readily propagated by the solid lung to the surface, and thus the phenomenon of tubular or bronchial breathing is heard—a sound similar to that which one may cause by simply blowing into a tube, or of which you may get a natural example by applying the stethoscope over the larynx and trachea in the neck.

And in bronchophony, it is as if the vocal sounds were generated in the air-passages; the voice seems to come from the lung. The fact is, that the vibrations excited at the vocal cords in the larynx are propagated along the walls of the bronchial tubes; and instead of being diffused through the soft, spongy lung, as in health, they are conducted in full force to the thoracic wall by its solidified portion.

You must not lose sight of the fact, that bronchial breathing and bronchophony may be present whenever a part of the lung is solid, whatever be the cause of the solidification, provided only a great portion of the bronchial tube leading to it be pervious. Thus it is that we often find these signs, when tubercular deposit has solidified a greater or less portion of lung. Sometimes pneumonia, running an insidious and chronic course, will make the lung solid, and develop these signs; or cancerous deposit may have been slowly taking place. To determine, then, the exact signification of these signs, you will have again to call to your aid the history of the symptoms of the case.

Again, you may have bronchial breathing when a slight pleuritic effusion has taken place; but such bronchial breathing will be accompanied, not with bronchophony, but with ægophony—a state of voice resonant and bronchial in its character, but rendered bleating by the interposition of a thin layer of fluid between the costal and pulmonary pleuræ. The existence of this modification of voice, and the simultaneous absence, or great diminution, of the usual vocal vibration when the hand is applied closely to the wall of the chest, will distinguish the bronchial breathing which is accompanied by pleuritic effusion from that which is due to the simple condensation of pneumonia.

Thus, then, the percussion, which we first practised on making our physical examination of the chest, directed us to the seat of lesion, and from it, together with the bronchial breathing, the bronchophony, the rusty expectoration, the local pain, the embarrassed breathing, and febrile disturbance, we diagnosed with confidence and certainty that the lower lobe of the right lung was in a state of inflammation, and had passed rapidly into hepatization. It is probable, too, that the inflammation of the lung was accompanied by some degree of pleurisy. Of this, however, we had no certainty, but three circumstances rendered it probable: first, because the local pain was sharper than it usually is in pneumonia; secondly, because on one day the bronchophony was decidedly ægophonic in character; and thirdly, because pneumonia (unless quite central and deep-seated) rarely occurs without some degree of pleurisy affecting the pulmonary layer of the pleura.

The portion of lung involved in the inflammation was rather more than one-third of the posterior part—the inferior third; and the inflammation extended half-way, or possibly two-thirds forward, towards the anterior surface of the lung. This was also the case in the second example of this disease which I shall bring before you to-day.

It is a remarkable feature in the clinical history of pneumonia, how prone it is to attack the lower part of the lung, and how much more frequently the posterior part is affected than the anterior, and how often the inflammation involves only a portion of one lung, and that portion not exceeding one-third or one-half, not often reaching to the anterior surface, and seldom extending through the entire lung; and, lastly, how, when both lungs are involved, they are symmetrically affected, so that you rarely find pneumonia in the base of one lung and the apex of another, but in the bases of both or the apices of both. I know of no satisfactory explanation of its partiality for the posterior and inferior part. Perhaps dependence of position, as likely to affect the circulation, may have something to do with it.

The extent of the inflammation, and, perhaps, in some degree, its position, ought always to be taken into account in forming a prognosis. If only a portion of one lobe is affected, recovery is much more frequent than the reverse, provided the treatment have not been of a too depleting nature; should the whole of one lobe be involved, or more than half the lung, the chances of recovery are much diminished; and should the inflammation engage the whole of one lung, the disease is very frequently fatal. When the pneumonia is seated in the upper lobes, the chances of recovery are much less than when it occupies the lower lobe. This is partly because when the disease attacks the upper lobe it is of a more asthenic nature, and partly also because this part is most apt to be affected in old persons. The clinical fact is highly deserving of your attention, and ought to exercise an influence upon your treatment.

*Treatment.*—Now, the treatment to which this patient, and the others whose cases I shall presently detail, were subjected, consisted in free counter-irritation by the application to the back and side, over the region of dulness, of flannels soaked in warm spirits of turpentine, which were kept on for half an hour. These stupes were applied at three several periods of the day, for the first three or four days. They excited considerable irritation and redness of the skin. A diaphoretic medicine was also administered, consisting chiefly of the liquor ammoniæ citratis, of which as much as six drachms was given every three or four hours; an occasional dose of a mild aperient medicine was given, and for food the patient was allowed at least a pint of beef tea daily, with milk and bread.

Let us see, then, the progress of the case under this treatment.

The patient, you will remember, came in on the 5th of January, which, reckoning from the date of the first occurrence of rigor and pain, was the third day of the disease.

On January 6th he was much the same as on his admission. Pulse, 96; respirations, 36.

On the 7th, a small patch of herpes was noticed at the right angle of the mouth, a phenomenon very common in pneumonia, and for some reason which I cannot explain, generally of favorable import. Pulse, 90; respirations, 36. A decided crepitation was heard at the end of each inspiration, although the breathing remained tubular. Sputa abundant, and less viscid.

On the 8th, pulse, 80; respirations, 40. The dulness on percussion was less extensive, and returning crepitation was now audible over the whole of the region of inflamed lung: fever less; tongue cleaning. The pain in the side being troublesome, ten leeches were applied.

January 9th (seventh day of the disease).—The pain was much relieved; bronchial breathing completely replaced by vesicular breathing and crepitation. Pulse, 96; respirations, 30.

On the 10th, the skin was moist and soft; sputa no longer rusty; crepitation audible on deep inspiration; and the voice slightly resonant beneath the scapula. Pulse, 60; respirations, 32.

On the 12th, the crepitation being still present, a blister was applied to the side; and on the 13th it was reported that the breathing was nearly pure, crepitation having almost disappeared. Pulse, 68; respirations, 28.

On the 17th, fourteen days after the first seizure, our patient was quite convalescent.

CASE LXXIV. (Vol. xxxii, p. 92.) The second case is that of a lad named Minns, aged 17, a waiter at a coffee-house. In his vocation, he is a good deal exposed to cold, but his health has always been very good. His illness commenced on February the 8th, with shivering, loss of appetite, cough, pain in the right side, and vomiting. In this patient the signs were of the same character as in the former, but were situated in the lower part of the right side. There was a similar acceleration of pulse and breathing. The percussion was dull over the lower part of the right lung behind, where also there was a total absence of vocal vibration. In other parts of the chest, the breathing was pure, and the percussion resonant.

Let me remark here, that in watching cases of acute chest disease, it is very important to note the frequency of the pulse and respirations, as furnishing one of the most useful guides in the progress of the case. When matters do not go right, you find the frequency of pulse and breathing increase from day to day, or remain stationary; but if, on the other hand, you find a gradual fall in the frequency of the pulse and breathing, you may feel sure that your patient is making satisfactory progress.

These physiological signs are not inferior in importance to any afforded by the patient in the course of his malady; and you may even trust to them alone, when they take a favorable course, to assure you, in the first instance, that the disease is not extending itself, and afterwards, that the inflammation is being resolved.

The diagnosis of this second case differed from that of the first, in this,—that, inasmuch as, in addition to the dulness on percussion, the usual vibrations excited by the voice were not felt when the hand was applied over the dull surface of the chest, there must have been fluid interposed between the lung and the pleura, to prevent their propagation to the walls of the chest; whence we concluded, that a slight pleuritic effusion accompanied the hepatization of the lung.

The progress of this case, under the same treatment as that applied to the first, was equally satisfactory.

Thus, he came in on the 10th of February, the third day of the disease; his pulse was then 118; his respirations, 38.

On the 11th, the pulse was 128; respirations, 34.

On the 12th (the fifth day of the disease) we found the pulse still high, 120; respirations, 40. There was, however, a manifest improvement in the physical signs. The vocal vibration was now to be felt, showing that the pleuritic effusion had been absorbed. Slight crepitation began to be audible near the base.

On the 13th, the pulse had fallen to 96, but the respirations were still as high as 42.

On the 14th, the seventh day of the disease, the pulse had fallen still lower, and, what was more important, the frequency of the breathing was reduced to 32; crepitation was now distinctly audible over the whole of the diseased portion of lung.

On the 15th, the crepitation had become much larger and moister, and a good deal of vesicular breathing was audible at the lower part of the lung; and on the 17th (the tenth day of the disease), all traces of crepitation had disappeared, and the dulness on percussion had diminished; the pulse was 78, and respirations 29.

Four days afterwards the patient left the hospital, quite restored to health.

Here, then, are two cases which I think you may take as examples of pneumonia, or more correctly of pleuro-pneumonia, of an average degree of severity. They by no means belonged to what is commonly called the asthenic or typhoid kind. The first, indeed, was distinctly sthenic; and the patient was a strong, athletic, muscular man, just such as one would suppose might be bled without much hesitation. The other patient was not of so vigorous a frame; but still, neither his constitution nor his symptoms were such as would have justified our regarding the case as, in the ordinary sense, asthenic. Yet you will observe, that, in the first case, the inflammation was fairly undergoing resolution on the seventh day of the disease, and on the eleventh day, the lung was in its natural condition: on the fourteenth day the patient was convalescent. In the second case, resolution was fairly established likewise on the seventh day of the disease, and on the fourth day of the treatment; and pure breathing was audible on the tenth day. A fortnight likewise was sufficient to restore this patient to complete convalescence. It is remarkable, that in both cases, the resolution should have taken place on the same day of the disease; but then it must be noted, that in both the treatment began on the same day, namely, the third from the seizure.

I have brought these cases before you as good illustrations of the progress of the disease under a mode of treatment which I have found most successful in a considerable number of cases, both in hospital and private practice. In this treatment, no attempt is made to cut short the disease: it is founded upon the observation of the way in which the disease is often spontaneously cured, through critical evacuations of sweat or urine, or of both, and consists, as I have already said, in an attempt to promote both these secretions.

But I am quite prepared to hear it objected, that such a treatment is really doing nothing but leaving the disease to take its own course. Well, and if that course be to recovery in a short time, and at no expense to the powers of the patient, can we adopt any plan better suited to him? I do not admit, however, that the frequent application to the chest of such counter-irritants as mustard or turpentine (three or four times a day), and large doses of the acetate or citrate of ammonia, and occasional purging, exercise no influence, either upon the whole system, or upon the local disorder. The drugs cause, undoubtedly,—and especially when the patient is kept in bed—free sweating or free diuresis, and often both; and it is quite consistent with all experience, that frequent counter-irritation exercises a beneficial influence on internal inflammations, and relieves pain.

I am not, indeed, aware of any mode of treatment which can be said, *bona fide*, to cut short the disease. The plan by bleeding and tartar emetic does not do so certainly. I have heard it stated, that large doses of digitalis will sometimes cut short pneumonia. Digitalis is an uncertain drug, not always possessing an equal amount of power, and sometimes not very controllable. Moreover, there are certain idiosyncrasies which do not bear the use of digitalis; nevertheless, it is not undeserving of trial in cases chosen with judgment. But generally, I believe we do more good in pursuing a simple plan of treatment, such as I have described, than in endeavoring to cut short the disease by remedies whose action is at most uncertain, but which may, now and then, do serious harm. By following the plan I have laid before you, if the patient dies, it will be rather from a negation of treatment than from any other cause: the treatment will prove unsuccessful, simply because the disease went on unchecked by it; and in such cases it is often a question if, by any known plan of treatment, such a result could have been averted.

Now, in the particular cases under consideration, a manifest check to the advance of morbid change took place immediately, the treatment was begun, and the signs of resolution followed very speedily; and I have already alluded to the curious fact, that the resolution in both cases was established on the same day of the disease, the treatment having likewise commenced on the same day—a fact which seems very much to indicate that the treatment had a good deal to do with the early resolution of the inflammation.

In estimating the value of this or that mode of treatment in any given disease, we should ascertain what are the natural tendencies of the malady—to recovery or to death. Is it a very fatal disease? When recovery takes place, what is the process? When death occurs, what are the immediate antecedents, and what its immediate causes?

These are points of clinical history upon which our returns are as yet far from being complete or exact. The numerical returns which we have respecting pneumonia are unsatisfactory, because in those returns all cases of the disease, whether asthenic or otherwise, and whatever be the extent of lung involved in the inflammation, are classed together. Suppose you were making inquiry respecting the results of treatment in cases of burns, how little information could you derive from numerical returns, if the cases were not classified according to the extent of surface involved in the burn! It is just so with pneumonia; we have as yet no classified returns; but, looking to general experience, and such numerical returns as we have, it may be stated that, as in burns, pneumonia is fatal in proportion to the extent of pulmonary surface involved; but that, in cases where one-fourth or a less portion of lung is inflamed, it has, on the whole, a very decided tendency to recovery. On the other hand, when the whole of one lung is involved, or when a considerable extent of both lungs is engaged, the tendency is as decidedly to a fatal result.

The fatality of pneumonia is also much influenced by the period at which the disease may have been detected, and some kind of medical treatment adopted, to the extent even of the mere adoption of the horizontal position in a warm bed. Thus, Grisolle's tables show that when the cases were brought under treatment within the first three days, only one-thirteenth died; but if not brought in before the fourth day, one-eighth died; if on the seventh, one-third; on the eighth, so large a proportion as one-half died. Age likewise exercises an important influence, and there can be no doubt that (excluding infancy, respecting which our facts are of the most unsatisfactory nature) the mortality increases with the age; and at the advanced periods of life, pneumonia must be regarded as a very fatal disease.

There are those who think, that when pneumonia affects the apex of the lung, it has a more fatal tendency than when it affects the base. I myself lean very much to this opinion, because in such cases (without referring to complication with tubercles) the disease is generally of the low or typhoid character; nay, under such circumstances, the pneumonia may be erratic, and like erysipelas, pass from one part to another of the same lung, and even to the opposite.

It may, indeed, I think, be laid down, that in all these cases, the pneumonia has a fatal tendency in proportion as it tends to exhaust, whether by its extent, or through too feeble powers of resistance on the part of the patient, or by exposure to cold and want of food, or by mental or bodily exertion during the early stages of the disease, or through some powerfully depressing influence connected with the original exciting cause of the disease, such as influenza, rheumatic fever, &c.

But if we take cases of pneumonia occurring in persons in the full vigor of life, and not involving a very large portion of the lung, and coming under treatment early, we may regard it as a disease of not very fatal tendency, but rather prone to get well, when the vital powers of the patient have not been too much depressed; and such cases will get well whatever be the treatment early adopted, provided no great error has been committed in either direction, either in that of reducing too much or of supporting too much, and it must be obvious to you, that that which most easily admits of correction is the latter. What we have to do in such cases is, to adopt the treatment which favors the shortest convalescence, and in the more severe cases we have to discover a mode of treatment which will promote the reparative process and uphold the powers of life.

The plan of treatment which has been recommended by some of our highest authorities, I need not tell you, is that by bleeding and tartar emetic. You bleed early from the arm, and if necessary you bleed a second or a third time; and if under this treatment resolution does not speedily take place, you bleed locally by leeches or by cupping, and likewise give tartar emetic more or less freely; to all which counter-irritation may be superadded in the more advanced stages. Mercury is also to be given freely, even to salivation, combined with opium.

I have had ample experience of this treatment; and I must confess it has given me so little satisfaction, that I have, for some years, ceased to adopt it; for, under this treatment, I have seen too many die; and when recovery has taken place, in too many instances has it been with a tedious, lengthened convalescence. Indeed, of all the fatal cases which it has fallen to my lot to witness, the great majority have been treated in this way; and in most of them antiphlogistic treatment had not been carried to an excessive or unreasonable extent.

In bleeding, the difficulty is to determine how much blood you may safely take away. Upon this point, I think, all who view the matter candidly must acknowledge that we have no satisfactory rule, notwithstanding the immense experience we have had of the practice. A loss of blood which scarcely makes an impression on one man will seriously reduce another; or a patient, who in a former illness has borne bleeding well, will suffer from it very much on a subsequent occasion; or losses of blood which were borne with impunity in one epidemic are injurious in another. All these are difficulties with which the greatest tact and judgment frequently find it impossible to cope. And, although in particular cases, relief may be afforded to certain symptoms by a timely bleeding, there is no doubt that in many it exercises no real influence in checking the progress of the disease; for, notwithstanding early bleeding, the lung becomes fully hepatized; nay, I would go so far as to say, that in some cases it favors hepatization by relaxing the bloodvessels and permitting a more ready transudation of the liquor sanguinis.

And as regards tartar emetic, I have long noticed that patients do best when the drug neither sickens nor purges. On this subject I am glad to fortify my own opinion, formed independently, by those of two such excellent authorities as the late Dr. Thomas Davies, and Dr. Watson. Dr. Watson, alluding to Dr. Davies, says: "He states, and this is accordant with my own experience of the remedy, that the tartar emetic always acts best when it produces no effect except upon the inflammation itself: *i. e.*, when it does not cause vomiting, or purging, or a general depression of the powers of the system." When, indeed, you can insure your patient against these effects of the drug, it is a very safe and useful remedy in pneumonia; but the difficulty is to limit its action in this way. This may be partially done, though not as regards its depressing effects, by combining opium with it, and the combination often exercises a favorable influence.

I had asked myself, why does this combination of tartar emetic and opium often tell so favorably in pneumonia? and the conclusion which I came to was this: because it tends very decidedly to promote sweating, and perhaps other excretions; and I was thus led to try drugs of a like tendency, such as liquor ammoniæ citratis or acetatis, in large and frequently repeated doses, which do not exercise such a depressing influence upon the patient.

In using these drugs, you must be careful to give full doses, four, six, or eight drachms, and to repeat them every three or four hours; and you must diligently apply, two or three times a day, or more frequently, counter-stimulants over a considerable extent of the surface of the chest, such as mustard, or flannels soaked in warm spirits of turpentine. With this you may often safely and with advantage combine the use of opium, and now and then a mild aperient will be necessary.

Under this treatment, deaths from pneumonia have become extremely rare among my cases. The fatal cases are those of patients who come under treatment being already far advanced in the disease, or in whom the disease has rapidly invaded a large surface of one or both lungs; but even such cases often do well under this treatment, combined with support and stimulants, if begun early.

In all cases, I am careful to give support from the first, in the shape of animal broths in small quantities at short intervals, and in most cases I give wine or brandy early, in a similar way, the dose being apportioned to the degree of depression of the nervous system.

In the decidedly typhoid cases, I need scarcely say, that the free use of stimulants is of essential importance; and it is often of immense advantage to give quinine freely, the special indication for this latter drug being profuse sweating.

Let me now direct your attention to a third case of pleuro-pneumonia. The attack was ushered in with symptoms of pleurisy, but afterwards pneumonia came on with well-marked signs. This is one way in which pneumonia manifests itself—the characteristic symptoms of the affection becoming developed upon an attack of pleurisy. I bring this case, also, before you, not so much to illustrate the history of the disease, as because it affords a good example of a case of rather severe pneumonia proceeding favorably, and terminating in a very satisfactory manner, under the plan of treatment which I have recommended to you.

CASE LXXV.[1] (Vol. xxxvii, p. 174.) The patient is a boy, in Sutherland ward, named W. Reddin, æt. 11, well-known, I hope, to many of you. On the 6th of the present month (November, 1852), he was seized with shivering and severe headache, which symptoms he attributed to having got a severe wetting a fortnight before.

He was admitted on the 10th, that is, four days after the occurrence of the shivering. When he came in he had a hot, dry skin, flushed face, and pain in the side; his pulse was 116, and the respirations 45. Besides these symptoms there was cough, with sore throat; but the patient did not expectorate.

On the 11th, the following physical signs were noted: Dulness on the right side posteriorly reaching as high as the scapula, with diminished vocal vibration, as compared with that found in the corresponding position on the opposite side. This led us to inquire whether fluid had not been effused into the cavity of the pleura in the situation above indicated. Upon carrying the hand down quite to the base of the right side of the chest, it was discovered that vocal vibration was totally absent,—tolerably certain evidence of the presence of fluid. Bronchial breathing was also audible behind, and a modified condition of bronchophony, which may be called "ægophonic bronchophony." From all these signs, then, we inferred that the lower third of the right lung was consolidated, as the result of pneumonia, and that a layer of fluid had been effused into the corresponding part of the pleural cavity, consequent on pleuritis in that position.

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[1] Reported by Mr. E. Liveing.

Upon listening higher up, crepitation, and a little higher still, pure vesicular breathing were audible. On the left side of the chest the breathing was puerile throughout the whole lung. Hence it was plain that we had to deal with a case of pleurisy accompanied with pneumonia, and consolidation of a certain portion of lung.

Such, then, was the condition of our patient on the 11th. Turpentine stupes were ordered to be applied to the affected part of the chest three times a day, as in the former cases, and half an ounce of the liquor ammoniæ acetatis was given every two hours; beef tea and milk were allowed as diet. Bleeding and all kinds of depletion were carefully avoided. The bowels were moderately acted upon. We were anxious to avoid pulling down the patient, the influence of the morbid process being quite enough for that purpose.

The treatment, then, was commenced on the 11th, or the fifth day of the disease. On the 12th, the physical signs were of much the same character. On the 13th (seventh day) the respirations had fallen from 44 to 30. On the 12th, the pulse was 90, and on the 13th, 84; while on the day of his admission it was 116, and on the day after, 112.

On the 13th, the skin was cool and moist, and the tongue clean. Vocal vibration had returned, and the bronchial breathing was less intense; but it had spread a little higher up, while at the same time crepitation was beginning to return in the lower part. With the returning crepitation, a slight pleural friction sound was noticed.

On the 14th the increased vocal vibration had entirely disappeared, and vesicular breathing was heard in the lower part, mixed with large crepitation. Upon placing the ear to the back of the chest, the vocal resonance was found to be natural, showing that the fluid had been absorbed. The pulse had fallen to 74, and the respiration to 24, and the patient was perspiring very freely.

Thus this patient passed through a severe inflammation of a considerable portion of one lung in nine days; but he might be said to have been safe in seven days. The morbid action began on the 6th with rigor, and reached its height in five days, probably in less time; and the time which elapsed from the adoption of the treatment up to resolution did not exceed three or four days, from the 11th to the 14th or 15th. On the 11th there was evidence of hepatization; on the 14th the hepatization had resolved; and since then the lung has been rapidly recovering its healthy condition. If now, in this case, I had taken blood, it would have been said that this good effect was to be attributed to the bleeding, and that the disease had been cut short. Under the remedies, however, which we have been adopting, the patient has done perfectly well, and we have the satisfaction of knowing that his constitution is unimpaired by any treatment to which he has been subjected by us.

Let me next allude to a very interesting point which has been noticed in these cases of pneumonia. It may now be looked upon as an established fact, that in this disease there is either a great diminution in the quantity of the chlorides, especially of the chloride of sodium, in the urine, or these salts are altogether absent from that fluid. This curious piece of chemical history was first pointed out by Redtenbacher, who records the results of his observations of it in eighty cases, in a paper in *Hibra's Zeitschrift der K. K. Gesellschaft der Aerzte.*

Dr. Beale has since confirmed the observations of Redtenbacher, and upon following up this subject, has made out, that while there is a deficiency of common salt in the urine, there is a corresponding excess in the fluid poured out into the lung; in fact, that the chloride appears to be drawn from different parts of the system to the inflamed lung.[1]

The amount of chloride in the urine gradually diminishes up to the period of hepatization, at which time, in the majority of cases, not a trace is to be detected. As soon as resolution commences, the chloride gradually reappears, until it reaches the normal quantity.

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[1] Medico-Chirurgical Transactions, vol. xxxv.

The method of testing for the presence of this chloride is so simple, that you should always ascertain for yourselves the accuracy of this statement whenever an opportunity occurs; indeed, your record of the case will not be complete unless it contains details respecting the quantity of the chlorides. All you have to do is, to add a few drops of nitric acid to a portion of the urine in a test tube, and then a few drops of a solution of nitrate of silver. If chloride be present, a dense white precipitate of chloride of silver, which is insoluble in acids, will fall. If it be altogether absent, no precipitate whatever will occur.

In the patients, Mills and Minns, the chlorides disappeared almost completely from the urine during the stage of hepatization, but returned to it on the reappearance of large crepitation, and on the resolution of the inflammation.

What appears somewhat contradictory of the above statement, in the case of our patient Reddin is, that on the 11th we tested this boy's urine, and found evidence of the presence of much chloride; but on the 12th there was a diminution, and on the 14th the quantity had again increased, so that in this case there was no total absence at any period; and from this circumstance alone we might infer that the hepatization of the lung was not very extensive. The disease began as pleurisy and passed into pneumonia, and it was not until hepatization took place, that the chloride in the urine diminished in quantity. It is quite possible that the precipitate was due to the presence of chloride of ammonium.

In uncomplicated pleurisy the chlorides are not affected. This we had recently full means of testing in the case of Mary Coley, who has just left the hospital. The rubbing sound was very loud and well marked. She was treated with opium, and large doses of liquor ammoniæ acetatis, and recovered rapidly. At no period of the case did we find any diminution of the quantity of chlorides in this patient's urine. In this case of pleurisy, then, the chlorides in the urine did not appear to be affected, while in pneumonia these salts undergo a marked diminution or disappear, and this is consonant with larger experience upon this

Pneumonia often comes on in connection with rheumatic fever, or in a highly rheumatic or gouty diathesis. Such cases bear ill the bleeding and tartar emetic system, but are particularly well suited for the plan which I now recommend to you, and in them opium may be freely used with very great advantage.

Most of you will recollect a well-marked case of this kind, of more than ordinary severity, in King's College ward, about six months ago, which was very successfully treated on this plan.

Case LXXVI. (Vol. xxxiv, p. 56.) The patient's name was Emma Keep; she was a pale, delicate-looking girl, seventeen years of age. From her history we learnt that she had enjoyed excellent health until about a year and a half previously, when she suffered from an attack of rheumatic fever, and was ill for six months. All the large joints were inflamed, and there appears to have been some affection of the heart, as she suffered from pain in the cardiac region, and oppression of breathing. As there was no evidence of valvular disease on her admission, it is probable that the pericardium was the part attacked, since when the lining membrane has suffered, abundant evidence of it is usually left, through the impairment of the valves, and the production of a bellows sound.

She was admitted on the 1st of November, 1851, and had then been ill only one day; all the principal joints were swollen and painful, and the usual symptom of profuse sweating was present. The treatment at once adopted was that by opium, alkalies, and moderate purgation, with the application of blisters to the affected joints.

On the 3d, the frequency of the pulse was increasing, a symptom which we always watch with anxiety.

On the 4th, the pulse was 116, and the respirations 34; the patient had an anxious expression of countenance, and complained of pain in her left side. Upon listening attentively in that situation, we were able to hear bronchial breathing, which extended over the lower part of the left lung behind, and there was decided dulness on percussion in the same region.

On the 5th, when listening to the heart, I detected, for the first time, a rubbing sound, accompanying both the systole and diastole: in fact, the characteristic to-and-fro rubbing of pericarditis.

On the 6th, she still complained of pain in the left side of the chest; the pericardial rubbing sound, the bronchial breathing and dull percussion continued, the latter extending over the inferior third of the left lung behind, when the patient was in a sitting posture; vocal vibration we found to be entirely absent over a space corresponding with the dulness, and at the junction of the inferior with the middle third of the chest on that side, there was that striking modification of voice called ægophony. Above the dull portion, and corresponding to the two upper thirds of the lung, the breathing was vesicular. At the base of the right lung behind there was some large crepitation.

The signs which I have just mentioned, are the most important indications of pleurisy with effusion of fluid; there could, therefore, be no difficulty in the diagnosis so far; but was this all? was there not also solidification of the base of the lung from pneumonic inflammation? I have no doubt that such was the case: the general condition of the patient, as well as the physical signs, favored that conclusion; but we are not able, in such a case as this, to derive the same certain evidence of the existence of pneumonia, which we usually do, from the physical signs, because a pleuritic effusion, compressing the lung, might alone be sufficient to produce them.

During the 7th, 8th, 9th, and 10th, the condition of the left side remained much the same, but the breathing at the base of the right lung acquired a decidedly bronchial character, crepitation being still audible. Rather later we were able to detect a pleural rubbing sound in the same situation. At this time the sputa were clear, but viscid and adherent to the sides of the containing vessel, and there was also present, in small quantity, the rusty mucus so characteristic of pneumonia.

On the 11th, the phenomena were as follows: "Pulse 116, respirations 36. Loud bronchial breathing was heard on the left side behind, and vocal vibration could not be detected below the angle of the scapula. There was slight crepitation quite at the base of the right lung, and bronchial breathing just above this." A slight pleuritic friction sound was also heard; vocal vibrations were present, though somewhat faint; the breathing at the upper part of the lung was clear.

There could be no doubt, then, that a certain amount of pleuro-pneumonia had been lighted up on the right side, in addition to that on the left; we had, in fact, to deal with a case of double pleuro-pneumonia and pericarditis. At the same time we were glad to find that the inflammation on the left side was not extending.

On the 12th, some amount of effusion into the right pleural sac seemed to have taken place, for the voice became ægophonic. Large crepitation and bronchial breathing were still heard. On the left side, the bronchial breathing was less marked; and on both sides respiration was clear, but feeble, to within two inches of the base. The pericardial rubbing sound, which had remained unaltered since its first occurrence, now ceased entirely.

For some days the rapidity of the pulse and breathing had been on the whole decreasing; and from this we argued favorably, although the case was a desperate one. On the 8th, the pulse and respiration were 120 and 40 respectively; on the 9th, 104 and 36. On the 10th, there was a slight increase in the frequency of the pulse: it rose to 110, but fell again the same evening to 100. On the 11th, the pulse was 116, and the breathing 36. This increase in the pulse was no doubt due to the accession of pneumonic inflammation on the right side. On the 12th the pulse fell to 96, and the respirations to 18.

On the 13th, there was a very decided improvement in the general condition of our patient. The pneumonia on the right side seemed resolving; the ægophony had disappeared; slight bronchial breathing was still audible at the base. On the left side, respiration was clear and vesicular over all the lung except quite at the base. Vocal vibrations were sensible to the hand to within two fingers' breadth of the lowermost part of the chest, where the voice was still ægophonic. Dulness on percussion remained; and this continuance of the dulness, after the other signs have disappeared, is a fact often observed; indeed, it never disappears with the same rapidity as the other signs, and it will often continue long after perfectly healthy respiration has been restored. I suppose that the pleura becomes thickened, or that some of the effused fibrinous matters are slow to become absorbed, or that the lung is not quickly restored to its normal density.

On the 14th, there was a return of the pleuritic friction sound on the right side, showing that the fluid had been absorbed, and that the surfaces of the pleura, rendered rough by the deposition of lymph, were opposed and in contact.

The articular affection had throughout been making slow but favorable progress, and by the 17th (the nineteenth day of the rheumatism, or the fourteenth of the chest affection) all the joints were free from pain, and the tongue clean. The heart sounds were perfectly normal, but feeble; the breathing over both lungs was clear and vesicular, except quite at the base, where, on the left side, ægophony was still heard, and on the right, a slight friction sound.

This patient was at first treated as we usually treat a case of rheumatic fever, with alkalies, opium, mild aperients, and blisters to the joints. But when the chest symptoms developed themselves, we then adopted a more free counter-irritation by the repeated application of turpentine stupes to the chest as often as every two hours. Blisters were also applied over the region of the heart.

Opium was also more freely exhibited. I deemed the use of opium to be more urgently needed in this case, in consequence of the rheumatic nature, and the great extent and complication of the chest disease. Usually it is not advisable to employ opium in large doses in the simpler forms of pneumonia, because it has some tendency to produce further congestion of the lungs and to depress the heart; but in such a case as this, where there was plenty of healthy lung, there was little to fear on that ground. Opium always acts beneficially in rheumatic fever; it relieves the pain, quiets the nervous system, and promotes the elimination of morbid matters by the cutaneous surface. We gave this girl as much as a grain and a half every three hours.

On the evening of the 11th, as matters were not progressing quite so favorably as we could wish, and there was evidence of a fresh inflammation of the right lung and pleura, I thought it advisable to order small doses of calomel. Half a grain was given every three hours, but altogether only three grains were administered, for on the following day the symptoms had so much improved, that I discontinued it, that we might not give this drug credit for having wrought changes with which probably it had nothing to do. Whether the favorable turn the case had taken was the result of these half-grain doses of mercury, I must leave you to judge; for myself, I must confess that I cannot believe it.

From this time our patient continued daily gaining strength; in a few days she was able to sit up, and was ordered middle diet and tonics, and on the 29th she left the hospital quite well.

Here then, in nine days (4th to the 13th) from the commencement of the symptoms, we find a case of double pneumonia, pleurisy, and pericarditis had run its course, and the subject of it been conducted safely towards convalescence.

Now ask yourselves, in a candid spirit, what was it which mainly promoted this speedy resolution of an inflammation so extensive, involving three such important organs? Was the process of cure a natural process, or was it the result of some influence exerted by the remedies administered upon the inflamed organs? Did they exercise some antidotal influence? or did they alter the blood in such a manner as to cut off from the inflamed part due supplies of what are called the products of inflammation?

Time would fail me were I to enter on the discussion of all these matters, however interesting and important. I must content myself with stating my own conviction, that the process of cure is a natural one, analogous to the union of wounds, or the healing of ulcers, and that a normal supply of blood both in quantity and quality is as necessary to the healing of the one as to the resolution of the other.

The following case will afford us another good illustration of the clinical history of rheumatic pleuro-pneumonia, in a severe form, and is the more worthy of your attention as it ended fatally. It was also rendered particularly interesting by the existence of symptoms which made the interpretation of the physical signs difficult, and led to a partially erroneous diagnosis.

Case LXXVII.[1] (Vol. xxxii, p. 78.) Patrick O'Reilly, a stout and well-nourished lad, aged 20, was admitted under my care on the 27th of January, 1851. His illness commenced five days before his admission with shivering and loss of appetite, followed by copious perspirations, pains in the joints, scanty and high-colored urine.

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[1] Reported by Mr. J. H. Sylvester.

On admission, his skin was hot and sweating, and he complained much of thirst; all the larger joints were swollen and painful; the tongue dry, and white in the centre; respiration a little embarrassed; pulse 108. On auscultation, the heart sounds were normal; but at the lower part of the left side of the chest behind, the expiration was bronchial, and the percussion over the same part dull.

The joints were wrapped in cotton wool, blisters were applied to some of them, and a turpentine stupe to the back.

On the 29th (the eighth day of the disease), he was on the whole much worse, although the articular affection was not quite so bad. Pulse 92, throbbing and intermittent; breathing 57, quick and labored; tongue dry and brown. He now suffered from thirst, and from a sense of oppression in the chest. The urine was scanty and high colored, loaded with red lithates, of specific gravity 1030, and containing a little albumen. There was cough and a slightly rusty mucous expectoration.

On examining the chest behind, two patches of bronchial breathing were found, one on either side, below the spines of the scapulæ; the expiration was prolonged, the voice bronchophonic, and the corresponding percussion dull. The base of the left lung behind was less dull on percussion; the breathing retained a tubular character, and there was some crepitation.

There was extended dulness on percussion over the cardiac region, a slight rubbing sound at the base, and a faint bellows sound at the apex.

The turpentine stupe was repeated, and a mustard plaster, to be followed by a blister, was applied over the heart. Five grains of nitrate of potass, with a grain of opium and a grain of ipecacuanha, were ordered to be taken every four hours.

During the next two days, January 30th and 31st (the ninth and tenth days of the disease), there was no alteration in the nature of the symptoms, but the patient's countenance began to wear an anxious expression; he lay continually on his back; the respiration became catching, the præcordial pain great, and the to-and-fro rubbing louder. Dulness on percussion and pleuritic rubbing were now detected at the base of both lungs behind.

The pupils were not contracted, despite of six grains of opium per diem.

Two grains of calomel were added to each opium pill. Blisters were applied to the back and front of the chest, and the turpentine stupe repeated. Mercurial and savin ointment was ordered to dress the blisters. A purgative enema was administered.

During the first week of February (the 11th and subsequent days) the nature of the symptoms and physical signs remained unchanged, but there were slight variations in their intensity, and some additional symptoms. Thus, signs of bronchial affection and pulmonary congestion, in the form of large crepitation and rhonchus, in the lower halves of both lungs, were noticed; and there was evidence of increased pleurisy in the loud, creaking, vibratory rubbing which could be heard and felt over the bases of both lungs. There appeared to be imperfect resolution of the two patches of dulness and bronchial breathing in the infra-spinous fossæ. The averages of the pulse and respirations were about 112 and 50 respectively. On the 3d there was drowsiness, with contraction of the pupils; on the 5th (the fifteenth day of the disease) the patient was slightly salivated.

Some anomalous symptoms were also present at this time, such as tympanitic resonance, metallic tinkling, and bulging of the wall of the chest, about the lower angle of the left scapula. These signs were difficult of interpretation at the time; they led for some time to the error in diagnosis of the existence of pneumothorax; but the phenomena of tympanitic percussion, with a metallic tubular breathing at the base of the left lung, were clearly traceable afterwards to the vicinity of an enormously enlarged stomach, pushing up the lung. Nothing could be more striking than the close resemblance between the phenomena afforded by this case, and those of an example of effusion of air and liquid into a portion of the pleural sac, and fistulous communication with the lung: the tympanitic splash on succussion, the tinkling of fluid dropping from one part of the cavity into fluid beneath, and even the amphoric blowing were present. But the tympanitic percussion sound was definitely limited by the line of a pushed-up diaphragm, whereas it should have varied its position, and spread over the whole left chest (at least posteriorly) had there been air in a non-adherent pleura. This point, if allowed its due weight, ought to have prevented the erroneous diagnosis.

On the 2d, or the twelfth day of the disease, wine was given for the first time to the extent of three ounces. On the 3d all the former medicines were discontinued, and a mixture, containing liquor ammoniæ acetatis with excess of ammonia and camphor mixture, substituted. On the 4th the wine was increased to four ounces; on the 5th, to six ounces, or half an ounce every two hours. Quina was now given in the day, and opium at night. On the 6th (sixteenth day) he had eight ounces of wine.

On the 8th of February (eighteenth day) some new symptoms manifested themselves. In the morning there were indications of pericardial effusion, in great pain and dyspnœa and increased extent of dulness over the cardiac region, with diminution of the to-and-fro rubbing and feebleness of the heart sounds. All medicine was omitted, and a blister applied.

In the evening expectoration of puriform matter in considerable quantity came on suddenly; the heart sounds were yet more muffled.

There was no important alteration for some days. The joints remained very painful, the pulse high, and also intermittent, the expectoration profuse.

On the 13th the patient became suddenly worse, his pulse more irregular; he now gasped for every breath, and died in about two hours, on the twenty-third day of the disease.

The post-mortem examination revealed a pericardium distended with bloody serum, except where it was firmly adherent to the surface of the left ventricle. The heart was coated with lymph and hypertrophied, and there was some aortic valvular disease. Recent lymph was found on the pleuræ, especially over the diaphragm. The left lung was compressed and pushed forward by the heart, and by an enormously enlarged stomach, and exhibited several patches of carnification. Both lungs were much congested; the bronchial membrane congested, and the tubes filled with muco-purulent fluid.

Here was a case of intense pericarditis which seemed to run its course wholly uninfluenced by any part of the treatment. Salivation, which took place on the 14th day, seemed to have no control, nor had opium freely administered from the first. Had a more vigorous antiphlogistic (so called) treatment been at first adopted, had free local or general bleeding been employed, and mercury given earlier and more freely, so as to have induced a rapid salivation, how would matters have stood? My experience of similar cases would lead me to say that, under such treatment in this particular instance, which *ab initio* was one of great extent and severity, the pericardial effusion would have been more rapid, the embarrassment of the heart greater, the partial resolution of the pneumonia which had taken place would not have occurred, the pulmonic solidification would have been much more extensive, and the fatal termination would have occurred on the 17th or 18th instead of the 23d day. Were I to treat such a case now, I should support the vital powers liberally and even largely from the first with broths and wine, or alcohol in some form, and employ opium and counter-irritation much more freely. The early adoption of a supporting treatment in such a case as this would have limited, rather than favored the extent of the inflammation. And even, had it failed in this important object, it would have given the patient a greater power of resistance to the depressing influence of disease, and promoted healing power, just as such treatment would have assisted the curative process in a large ulcer, or in an open stump.

## ADDENDUM TO LECTURE X.

The three following cases are extracted from the *Medical Times* for February 19th, 1859, and reprinted here as an important *addendum* to the previous lecture, and an illustration of the practice there recommended in the hands of three different physicians. The third case is one of typhoid fever of which pneumonia was a prominent symptom.

Case LXXVIII. James Jackson, aged 11, was admitted into King's College Hospital, December 29th, 1858. (Vol. lvii, p. 181.) It appeared that on Christmas eve the boy had eaten a number of hollyberries, and was afterwards seized with vomiting and violent convulsions. The exhaustion which followed was so alarming that his life was despaired of; but after taking a considerable quantity of wine, he began to revive. A day or two afterwards he was attacked with pneumonia, for which he was admitted under Dr. Todd's care.

When his chest was examined, it was found to be malformed —"pigeon-breasted," as it is commonly called. There was dulness on percussion over the whole right lung behind, and for four or five inches beneath the clavicle in front. Over the dull region bronchial breathing and bronchophony were distinctly audible. His skin was hot and dry: the tongue brown and parched. Pulse 116; respirations 38. He was ordered a mixture containing liquor ammoniæ acetatis, with excess of carbonate of ammonia and chloric ether, turpentine stupes to the chest night and morning, and half an ounce of brandy every two hours.

The report of the next day, December 30th, is as follows: "He coughs occasionally, but swallows the expectoration. Has rather a drowsy, intoxicated appearance, and comprehends with difficulty what is said to him. Urine, specific gravity 1017; the quantity of chlorides greatly diminished. Pulse 128; respirations 48."

On the 31st, the pulse was 123, the respirations 46. He was then expectorating viscid brownish sputa. The drowsiness continued. Chlorides were all but absent from the urine. On the 2d of January the pulse and respirations were 112 and 44 respectively. As his skin continued very hot and dry, the dose of liquor ammoniæ acetatis was increased.

On the 3d, the ninth day of the disease and the sixth of treatment, resolution appeared to be commencing. Bronchial breathing and bronchophony were persistent over the right lung behind, but in front loose crepitation, mixed with bronchial breathing, was audible. The quantity of chlorides in the urine was slightly increased. Pulse 104; respirations 40.

By the 5th, there was a very great improvement; the pulse had fallen to 90, and the respirations to 24; the breathing was much quieter, and crepitation was audible behind as well as in front. His cough continued troublesome, and he slept badly. The following day the quantity of chlorides in the urine had greatly increased.

On the 10th, the pulse was 70, and the respirations 24. The dulness over the right lung, both in front and behind, had diminished, and bronchial breathing and bronchophony were disappearing; loose crepitation remained.

On the 12th, the eighteenth day of the disease, and fifteenth of treatment, the resolution was nearly complete; the dulness remaining over the right lung was very slight, and the chlorides had returned in normal quantity to the urine. There was still increased vocal resonance, with rhonchus, sibilus, and loose crepitation. Pulse 72; respirations 24. Turpentine stupes discontinued.

On the 14th, the frequency of the pulse and respirations had slightly increased; they were 88 and 28. The patient's face was flushed, and there was increased dulness, with crepitation, over the right lung. The turpentine stupes were resumed. This unfavorable change was very transient, and by the 17th the pulse and respirations were again 72 and 24, and the dulness was confined to the base of the right lung behind.

By the 19th, a small secondary abscess had formed on the left cheek, and was opened. The pulse rose to 100, the respirations to 32. He was ordered three grains of citrate of iron three times a day.

On the 24th, the abscess had healed, and he was reported convalescent. He was discharged on the 27th.

*Tabular View of Pulse and Respiration.*

| Date. | Pulse. | Resp. | Date. | Pulse. | Resp. |
|---|---|---|---|---|---|
| Dec. 29, | 116 | 38 | Jan. 6, | 90 | 24 |
| " 30, | 128 | 48 | " 10, | 70 | 24 |
| " 31, | 123 | 46 | " 12, | 72 | 24 |
| Jan. 2, | 112 | 44 | " 14, | 88 | 28 |
| " 3, | 104 | 40 | " 17, | 72 | 24 |
| " 5, | 90 | 24 | " 19, | 100 | 32 |

Case LXXIX. J. D., aged 6, was admitted into King's College Hospital, under the care of Dr. Budd, on December 2, 1858, in a state very closely resembling delirium tremens. She had been placed out to nurse for some time previously, and her mother could give no account of her illness, except that on November 29th, she first complained of feeling unwell. She stated, too, that her child had been fond of spirits, but, as far as she was aware, had never had more than two or three teaspoonfuls occasionally.

On admission: face slightly flushed, with anxious expression. She is in a constant tremor, occasionally delirious, moaning and crying out, and now and then asking for beer and gin; frightened by slight causes. When an attempt is made to raise her in the bed she cries out; sleeps ill; has frontal pain, and the forehead is very hot. Breathing rather labored, Chest and heart sounds normal. Skin hot and feverish. Urine loaded with lithates; contains no albumen. Pulse 136; respirations 36.

℞. Tr. opii, ♏j; sp. æth. chlor. ♏ij; aquæ ℥j; statim sumend.

℞. Sp. ammon. arom. ♏x; mist. camph. ℥j; ter die sumend.

Dec. 6.—Pulse 136; respiration 52. She lies in a very drowsy state, but has lost the constant tremor which she had on admission. Has been wandering a good deal, and yesterday she was delirious. On examining the chest behind, there is dulness on the left side about the spine of the scapula, with bronchophony and bronchial breathing. Large and small crepitation are audible over the whole of the left lung behind. Tongue coated and dry. She passes her urine under her.

℞. Sp. ammon. arom. ♏xx; sp. æth. chlor. ♏v; mist acaciæ ℥ss; 4ta quâq. hor. sum.

Turpentine stupes to chest night and morning. Wine ʒij every hour.

8th.—Pulse 100; respirations 40. Breathing less labored. Still lies in a drowsy state, and is with difficulty roused. No change is perceptible in the condition of the left lung. Tongue coated. Urine turbid, pale, of neutral reaction; contains no albumen, and the chlorides are entirely absent.

9th.—Pulse 100; respirations 40. Crepitation is now heard over the front of the chest on the left side. Bowels rather relaxed.

10th.—Pulse 100; respirations 36. Face rather flushed; looks more lively. Tongue clean and moist. Takes her food well. Bowels moved once. The physical signs are the same.

11th.—Pulse 88; respirations 28. The crepitation has disappeared from the front of the chest, but bronchial breathing and bronchophony are still audible behind. Urine of normal color; the chlorides have returned, and are in the ordinary proportion.

13th.—Going on favorably. Omit wine. Pulse 80; respirations 26.

14th.—Respirations 24; pulse 76, weak and intermittent, corresponding with the action of the heart. She looks bright and cheerful, and feels much better. Slight tubular breathing is still heard about the inferior angle of the left scapula. Appetite very good.

17th.—Pulse 64; respirations 24. Looks rather pale and weak.

20th.—Gets up a little every day. Looks much better, and is getting stronger.

23d.—Convalescent.

31st.—Discharged cured.

*Tabular View of Pulse and Respiration.*

| Date. | Pulse. | Resp. | Date. | Pulse. | Resp. |
|---|---|---|---|---|---|
| Dec. 2, . . . . | 136 | 36 | Dec. 11, . . . . | 88 | 28 |
| " 6, . . . . | 136 | 52 | " 13, . . . . | 80 | 26 |
| " 8, . . . . | 100 | 40 | " 14, . . . . | 76 | 24 |
| " 9, . . . . | 100 | 40 | " 17, . . . . | 64 | 24 |
| " 10, . . . . | 100 | 36 | | | |

CASE LXXX. M. K., aged 36, resident in London for the last twenty-six years, was admitted into King's College Hospital, under the care of Dr. Johnson, on January 13th, 1859. Has been a nurse in the Fever Hospital for the past two years. Says she never had a day's illness in her life before. The present attack came on about three weeks ago, with pains in all the limbs, and rigors. She answers questions in a very confused manner. Bowels constipated; tongue coated with a dark brown fur; sordes on the teeth. There is considerable engorgement of the right lung posteriorly and inferiorly. The expectoration is scanty, and very viscid. The abdomen is covered with a large number of rose-colored spots, but there is no tenderness on pressure. Urine acid, contains no albumen. Pulse 120; respirations 36.

℞. Ammon. ses. carb. gr. v.; sp. æth. chlor. ♏x; mist. acaciæ ℥jss; 4ta quâq. hor. sum. Turpentine stupes night and morning. Brandy, ℥ss every two hours. 9.30 P.M., pulse 132; respirations 40.

Jan. 14th.—Continues much the same. There is rhonchus and sibilus over both lungs posteriorly. Urine decidedly deficient in chlorides. Ordered—Brandy, ℥ss every hour; adde sing. dos. mist. liq. ammon. acet. ʒiij. Pulse 120; respirations 32.

15th.—Pulse 124; respirations 32. Rhonchus and sibilus over both lungs, anteriorly and posteriorly: over the right back there is slight dulness on percussion. Bowels moved once yesterday.

17th.—Pulse 104; respirations 33. Bronchial breathing over the right lung posteriorly, and the dulness has increased. Tongue clean and red at the edges, and coated with a brown fur in the centre. Expectoration viscid, and of a rusty color.

20th.—Pulse 88; respirations 36. Dulness and bronchial breathing diminishing over right back, and loose crepitation is now audible there. Rhonchus and sibilus heard over both lungs anteriorly and posteriorly. Expectoration copious and rust-colored.

22d.—Pulse 88; respirations 34. Loose crepitation alone is audible over the right lung behind. Cough much less troublesome; expectoration free, copious, and losing its brown color.

27th.—Nothing but rhonchus and sibilus heard over the chest; expectoration quite clear. Patient feels well, and suffers only from weakness.

℞. Quinæ disulph. gr. j; acid. sulph. dil. ♏v; aquæ ℥jss t. d. s.

Feb. 1.—Chest sounds normal; the patient is now convalescent.

*Tabular View of Pulse and Respiration.*

| Date. | Pulse. | Resp. | Date. | Pulse. | Resp. |
|---|---|---|---|---|---|
| Jan. 13, | 120 | 36 | Jan. 17, | 104 | 33 |
| 9·30 P.M., | 132 | 40 | “ 20, | 88 | 36 |
| Jan. 14, | 123 | 22 | “ 22, | 88 | 34 |
| “ 15, | 124 | 32 | | | |

## LECTURE XI.

### *On Pneumonia.*

Gentlemen,—The purport of a clinical lecture is to bring more immediately before you the prominent points of one or more cases, which may be actually under observation, or may have been recently so. Whilst the more immediate aim of this kind of instruction is to teach you the clinical history of disease by examples, it also tends to assist and direct you in making observations and in keeping records of your observations with such fulness and accuracy, that they may be useful hereafter, not only to yourselves, but also to others.

A diligent and accurate chronicler of the day by day phenomena and changes which occur in particular cases of disease, is, in his way, a highly important contributor to the cultivation of medical science. He supplies the material out of which may be framed, hereafter, the most valuable additions to our views of pathology and treatment.

> "Sicut
> Parvula (nam exemplo est) magni formica laboris
> Ore trahit quodcunque potest, atque addit acervo
> Quem struit, haud ignara ac non incauta futuri."

I am sure that it would be impossible for me to thank too cordially, or commend too highly, the gentlemen who, acting as my clinical clerks, have from time to time kept accurate records of such cases as I have had to treat in this hospital; on the other hand, those who have neglected that important duty, and have been slovenly and careless in their records, would be the first to regret their want of attention on finding how utterly useless and fruitless their records prove to be, for any purpose, either of learning or teaching.

These remarks have been suggested by my having had to look into the records of my cases with reference to the present and one or two succeeding lectures. You know that, now and then, I like to give a group of lectures on some particular malady or class of maladies, and to illustrate them by reference to cases recorded in former years, as well as by those recently under observation. The records of former years are, therefore, of great importance to me, and I am obliged to lay my case-books freely under contribution. During the present session, I propose to direct your attention a good deal to diseases of the lungs.

Although there is no necessary connection between the acute and the chronic forms of disease of all the great internal organs in general, and of the lungs in particular, one naturally, in discussing the diseases of an organ or tissue, begins with that of simple inflammation; and it forms a very fitting introduction to the study of pulmonary diseases in particular, as tending to familiarize you with some of the more important signs and symptoms of those diseases, and to teach you to what an extent the anatomical characters of the lung may undergo alteration, and yet the organ may ultimately perfectly recover itself. Inflammation of the lung-tissue, too, is the most formidable, although not the most fatal, of the acute diseases of these organs, and under certain circumstances leads to the destruction and breaking down of the lung-substance in a very short time.

It so happens, that during the last week, an excellent example of *simple inflammation of the lung* has been under our treatment in the hospital, in the case of a little boy in Rose ward, and this has afforded us a good opportunity of studying the disease. I need, therefore, make no further remarks on the propriety of my taking first this important subject, but will proceed at once to discuss the clinical history of the disease.

Let me commence, then, with noticing the various circumstances under which PNEUMONIA is met with in practice; in other words, let me state what are its clinical varieties. They are these:—

1st. *Simple pneumonia* (of which you have an example in the case now under observation, to which I shall have to direct your attention presently), that is to say, pneumonia uncomplicated with disease of any other organ, and occurring in a subject who possesses no marked peculiarity of constitution. But pneumonia is most frequently complicated with inflammation of the pleura, and, indeed, I suspect that we seldom meet with a case of this disease in which this membrane does not participate more or less, in the inflammatory condition; for the pleura, as most of you are aware, is a delicate film of membrane covering the lung, and receiving its nutrition from the pulmonary bloodvessels; and it is, I apprehend, scarcely possible for the superficial lung structure to become inflamed, without this membrane, which is so intimately connected with it, being also involved in the morbid process. Hence we have the term "*pleuro-pneumonia,*" which is applied to this inflammatory condition of the pleura and lung.

*Simple pneumonia* is very rare in another sense also, that is, in its freedom from complication with or dependence on some peculiarity of constitution. To make this clearer to you, let me take an illustration. If two men, A. and B., both in good health, be exposed to some noxious influence, cold for example, at the same time, and for the same period, A. will get a severe attack of pneumonia, and B. will not. Now at first sight one can scarcely conceive why the pneumonia should attack the one and not the other, for they were both apparently equally well at the time of the exposure to cold; but, if we carefully examine into the previous history of these individuals, we shall find that A. is of a gouty or strumous constitution, or has some peculiarity of diathesis which B. does not possess, and it is by reason of this that A. is seized with pneumonia when subjected to the noxious influence, which produces no such injurious effect on B. No doubt, there are few cases in which there is not some peculiarity of constitution which may determine more or less the access of pneumonia, and influence its duration and mode of termination; still when inflammation of the lung occurs without any concurrent disease, and without any marked peculiarity of diathesis, it is convenient to call it "*simple pneumonia.*"

2d. Pneumonia complicated with *acute gout*, or with *rheumatic fever*, or associated with a decided gouty or rheumatic diathesis. This form of pneumonia is of very common occurrence, especially in aggravated states of the gouty or rheumatic constitution. It sometimes comes on in the middle of an attack of acute gout or rheumatic fever; at other times it ushers in the attack; while, in a third set of cases, it follows the constitutional malady,—the one or other of these, as the case may be, passing away, and leaving behind it inflammation of the lung.

3d. *Strumous pneumonia; i. e.*, pneumonia connected with the development of tubercles in the lungs, or occurring in subjects of a strumous constitution, without any evidence of tubercular deposits in these organs. This latter form is frequently met with in strumous children, and is often, I suspect, mistaken for *phthisis*.

4th. *Typhoid pneumonia;* by which I mean, inflammation of the lung coming on in a low state of the system, and associated with a series of typhoid symptoms, such as great prostration, a brown tongue, and a languid and feeble condition of the circulation; or you may have the disease specially connected with either typhoid or typhus fever. (Case lxxx.)

5th. *Traumatic pneumonia; i. e.*, pneumonia succeeding and consequent on injuries to the chest, or following severe surgical operations.

These are the distinct and undoubted clinical varieties of pneumonia, which you must be prepared to deal with in practice.

Some physicians speak of *lobular pneumonia* as occurring in young children, and associated with extensive bronchial inflammation, or with hooping-cough. But I believe that the condition of lung which in these cases has been attributed to inflammation, is not so, but is merely a state of carnification, due to the exhaustion of air from parts of the lung by the excessive expiratory efforts which occur in these cases.

Let me now explain to you in what pneumonia consists, and what are the anatomical characters of the disease.

Pneumonia may be defined to be "that condition of lung which leads to the formation of a plastic deposit in the cavities of the air-cells, which plastic material, by filling up the interior of these cells, and the finest bronchial tubes, consolidates the previously soft and crepitant pulmonary tissue." This plastic substance is probably of an albumino-fibrinous nature, formed, no doubt, from the liquor sanguinis which exudes from the pulmonary bloodvessels. These vessels project, as you are aware, uncovered, from the walls of the air-cells, and are, consequently, very favorably situated to permit transudation through their coats. This exudation by its coagulation very quickly consolidates the lung, and, being mixed, more or less, with the coloring-matter of the blood, gives that organ, when cut into, very much the consistence and color of liver. I have known good anatomists mistake for liver a piece of lung in a state of *red hepatization*. This condition, which as I just now said, is simply produced by the effusion of a plastic material entangling blood particles, and the consequent consolidation of the air-cells and finest bronchial tubes, converting the previously soft and spongy pulmonary tissue into a solid mass, is the only *certain* indication, with which I am acquainted, of pneumonia having existed during life.

This state of red hepatization is, nevertheless, preceded by one in which the blood is delayed in that portion of lung about to become hepatized, and, very probably also attracted to it in increased quantity—a state, in fact, of what has been called *active congestion;* in this consists the first stage of pneumonia.

The real nature of the organic changes which constitute this state of active congestion may be thus explained. Some matter introduced either through the bronchial tubes with the inspired air, or through the blood, irritates a certain portion of the lung; in other words, disturbs its nervous influence, and deranges its nutrition. The immediate result of this nervous derangement is an increased action of the heart, a dilated and enfeebled state of the contractile wall of the finest capillaries, which offer no resistance to the flow of blood to that part, and allow it to accumulate there in greatly increased quantity. This relaxed state of the capillary wall increases the size of its pores, and allows a freer transudation of liquor sanguinis than takes place in health, and giving rise to those subsequent changes which produce the condition of red hepatization.

It is not possible, in the present state of our knowledge, to lay down any anatomical characters by which this first stage of pneumonia can be positively determined after death. The existence of a patch of reddened lung, with distended capillary vessels, the pulmonary tissue being slightly œdematous and increased in density, and the borders of the patch gradually shading off into healthy tissue, would lead me strongly to suspect that inflammation had existed during life, and that that portion of the lung would have speedily passed into red hepatization.

But it is very rare for patients to die in this early stage of pneumonia.

Sometimes active congestion will exist in connection with acute bronchitis. In such a case there will be great intensity of color in the mucous membrane of the bronchial tubes, large and small, and the congestion will involve the greater part, or the whole of one lung, and very frequently of both lungs. But there are no distinct anatomical characters attaching to such a congestion, which would enable the anatomist to predicate of a portion of lung placed before him, this is the congestion of bronchitis, and not of pneumonia, or *vice versa.*

Passive congestion of the lung may be induced by anything which impedes the passage of the blood through the pulmonary veins, such as diseased heart, or the pressure of a tumor or aneurism on the root of the lung, or on one or more of the pulmonary veins. In asthma and its resultant emphysema, the lung becomes congested in consequence of the imperfect aeration of the blood, and the failure of the proper capillary force, which is so important an aid to the pulmonary circulation. So also in asphyxia, congestion is due mainly to imperfect aeration, and the consequent failure of the same important aid to the capillary circulation.

Whatever delays the flow of the circulating fluid, throws back the blood on the left auricle, and thereby impedes its passage through the pulmonary veins, will produce congestion of the lungs. In various low diseases, which involve a prolonged decubitus on the back, a considerable amount of pulmonary congestion is invariably found as the result of gravitation. In typhus, measles, pyæmia, and other diseases due to the influence of an animal poison, the pulmonary congestion, which is so apt to occur in them, is partly passive and partly active, owing to the irritant action of the animal poison on the bronchial membrane.

You will see, therefore, that the fact of a lung being gorged with blood, which flows freely from the divided vessels when it has been cut into, is no proof of the existence of inflammation in it during life. This point can only be determined (for the first stage) when such anatomical characters as I have described follow certain symptoms and physical signs; and it is one of those cases which indicate how valueless is morbid anatomy, if studied without the light of careful clinical observation.

In the commencement of the second stage of pneumonia or that of *red hepatization*, the plastic material, which is poured out in the air-cells and finest bronchial tubes, takes the shape of these cavities, and is sometimes in part spat up, giving rise to *casts* of the cells and finer air-tubes, which, when subjected to microscopic examination, are found to be analogous to those which are formed in the kidneys in consequence of irritation in the renal tubules.

It is remarkable how rapidly the first stage of pneumonia, or that of active congestion passes into that of red hepatization; in other words, how quickly a lung becomes hepatized. Laennec asserts that a *fine crepitant râle* may be heard on listening to a lung in the first stage of pneumonia, or that of active congestion, and that this is speedily succeeded by the signs of red hepatization; but in the whole course of my experience, I have had but very few opportunities of hearing this râle ushering in the stage of red hepatization, and which was considered by Laennec as pathognomonic of pneumonia. This is explained partly by the rapid transition to which I have referred, and partly by the fact to which Skoda has directed attention, that this condition of lung is in general immediately preceded merely by catarrhal râles in the bronchial tubes, and not, as believed by many, by a peculiar kind of fine crepitation.

The second stage of pneumonia quickly passes into the third, or that of *gray hepatization;* the red color of the lung disappears, but the organ remains solid, and when cut into, looks not unlike a piece of gray granite. This stage, too, like the second, in a very short time passes into one in which more or less of pus is infiltrated into the substance of the lung, and the matter scraped off the cut surface, exhibits, under the microscope, great numbers of cells, having the distinctive characters of those of pus. When pneumonia has advanced even to this stage, the lung may perfectly recover itself, the patient expectorating a large quantity of pus; indeed, so large is the quantity sometimes spat up in these cases, that one is often led to fear that an abscess has formed in the lung. But, curious to relate, a true pneumonic abscess is one of the rarest things which we meet with in morbid anatomy; and I cannot now call to mind more than three instances of this kind that have fallen under my own notice. This fact of the extremely rare occurrence of abscess of the lung following pneumonia is a point upon which authors are singularly unanimous. If death occurs in pneumonia, it is most likely to take place in the stage of purulent infiltration; but even then the patient may get quite well by the free discharge of the purulent matter. Recovery may take place in any stage of pneumonia. It generally follows red hepatization, and nothing is more extraordinary than the rapidity with which a lung will pass from the state of red hepatization to that of health. The way in which this change occurs is probably this: a fresh effusion is poured out, which consisting chiefly of serum, dissolves the plastic material with which the cells and finer air-tubes were previously blocked up; a portion of this is re-absorbed, while the remainder is quickly expectorated, the dilated capillaries gradually resume their natural size, and the lung returns to its normal condition. In the case which I shall presently bring before you, the rapidity of this change was very striking. When recovery takes place in the stage of gray hepatization, it is much more slow; and the same obtains in the stage of purulent infiltration, as I just now observed.

Case LXXXI. (Vol. xl, p. 130.) Such, then, are the preliminary remarks with which I have thought it expedient to bring under your notice the case of a boy named Everitt, now in Rose ward. He is a fair-complexioned lad, of a strumous diathesis, and his antecedents indicate that he has been much in the hospital, for "when two years of age he had hooping-cough, and from this he suffered greatly the following twelve months." He "wasted away;" and so severe and trying was this malady, that "he was given up by all the doctors who attended him." After the disease had lasted twelve months, it left him; that is to say, he ceased to hoop, but the cough remained; and from that time to the present he has been a poor, weakly child, always having a cough and spitting, the matters expectorated being usually "thick yellow and greenish phlegm." In February last (1853), he had an attack of scarlatina, for which he was three months in the hospital under my care. The fever was followed by a slight degree of general dropsy, and his urine contained at that time a considerable quantity of blood (free, and also in casts of small diameter), with a few epithelial casts, but with very little desquamated renal epithelium; and it was also slightly albuminous, but probably not more so than would be due to the amount of blood present—all these signs indicating, as you are aware, an irritated and inflamed condition of the kidneys.

The pneumonia in this boy came on in the regular way; indeed, the case affords a good example of the mode in which this disease usually begins. "His present illness," says the report, "commenced on the 19th inst. (October, 1853), when he was taken with running at the nose, shivering (distinct rigors), a *stitch* in the left side, and vomiting." All these symptoms are of nearly constant occurrence in the development of a well-marked case of pneumonia, except the vomiting, which is rare.

"On the evening of that day the shivering left him, and he became very hot and feverish;" and I may here remark that this heat of skin is looked upon by some physicians as highly characteristic of pneumonia. "On the following day he appeared to be getting worse, his breath began to get very short, and the vomiting and stitch continued, and on the 21st he became an out-patient of the hospital;" but of the nature of the treatment upon which he was put at this time there is no record in the notes of the case. "All this time his cough, which was usually very troublesome, seemed to get better—he coughed very little, and did not spit up anything—it (the sputum) seemed too thick to get up." From the characters of the sputa we derive, as you know, considerable aid in forming our diagnosis in cases of pneumonia; and from the patient's own description of facts we oftentimes receive very useful hints. This very circumstance of the sputa "being too thick to get up" would at once lead a person, accustomed to think on this subject, to suspect the existence of inflammation of the lung, as pneumonic sputa are very thick and viscid, and cling to the sides of the vessel in which they are contained, so as not to fall out even when it is inverted. Indeed, so very adhesive is the pneumonic expectoration, that it sticks to the sides of the bronchial tubes, trachea, and larynx, this property being due, no doubt, to its containing a large quantity of the plastic material of which I spoke at the commencement of the lecture; and it is in this thick and viscid mucus that we sometimes find those little "casts" of the finer bronchial tubes, which are almost pathognomonically characteristic of the sputa in pneumonia. Another character peculiar to the matters expectorated in pneumonia is their color, being tinged by an intimate admixture of more or less of the coloring-matter of the blood, which usually gives them a *rusty* hue. Sometimes, however, this rust-color is not so distinct; and occasionally, in genuine cases of pneumonia, the sputa have, instead of it, a bilious tint.

After the 21st inst., we have no distinct history of the case until the patient's admission into the hospital on the 25th. "He appeared," say the notes, "to get worse, his breathing became more uneasy, and the fever increased;" and on his admission on the 25th, his condition is thus described: "A weakly, delicate-looking, fair-complexioned lad, with a flushed face, and considerable shortness of breath; P. 120; R. 50; skin hot, dryish; tongue coated with a whitish fur, moist; no appetite; great thirst; complains of pain in the left side, over the region of the left lung, and of cough, but does not expectorate at all, or if he does, he swallows the sputa, but of this last there is no decided evidence; the left side of the chest, especially at the lower part, hardly expands so much on inspiration as the right." From all these sysmptoms—the hot skin, the shortness of the breath, the pain localized in one spot on the left side, that side of the chest not expanding on inspiration so much as the other, &c., one would at once be led to suspect the existence of pneumonia.

By *percussion* and *auscultation*, we obtained further signs which left no doubt as to the nature of the disease. "On percussion there was dulness over the lower part of the left lung posteriorly (the region in which the pain was felt); the dulness was not so complete as that which would be produced by the presence of fluid in the pleural cavity, but had rather the characters which would be due to a nearly solid viscus including some amount of air." We may suppose that though the air-cells were filled with the plastic material, the larger bronchial tubes, passing through the substance of the lung, contained air; and I mention this circumstance particularly, because I wish you to bear in mind that the term "dull," as applied to the sound elicited by percussion, is a *generic* one, and may be divided into several special kinds, each differing from the other in a marked manner, though all of them are essentially *dull*. Thus, there is a remarkable difference between the character of the *dull* percussion sound which is produced by an inflammatory consolidation of lung, and that which is the result of pleuro-pneumonia with the presence of a *small* quantity of fluid in the pleural cavity; and the *dull* sounds, produced by these two conditions respectively, differ considerably in their characters from that which is due to the existence of a *large* amount of fluid in the cavity of the chest. "The percussion-sound over the right lung was quite clear."

Another sign, which naturally follows upon this one of percussion, is the effect of the voice on the walls of the chest; if the lung be healthy, vibrations are felt, but if fluid intervene between the lung and the costal pleura, no such vibrations can be distinguished; while if the lung be solid, but the larger bronchial tubes are permeated by air and communicate freely with the trachea, vocal vibrations, sensible to the hand, will still exist and sometimes in an exaggerated form. In our patient vocal vibrations "were about equal over both lungs—if any difference, slightly stronger over the left." Here, then, was an additional reason for not attributing the dulness on percussion to the presence of fluid in the pleural sac; for, had that been the case, no vocal vibrations would have been detected. By comparing the vocal vibrations on both sides, we found that they were slightly stronger over the left lung; and this brings to mind a point on which I wish to dwell for an instant. In some cases of pneumonia the vocal vibrations are most distinct over the diseased lung, in others they are best marked over the sound one, while, in a third set of cases, they are altogether absent over the inflamed organ. This difference in the degree of the vibrations of the thoracic walls produced by the voice, seems to me to depend on the condition of the bronchial tubes; the more freely the air passes through these tubes, the more distinct will be the vocal vibrations, and, conversely, the more plugged up these tubes are, the less perfectly will the chest vibrate.

On listening to the chest, we found the phenomenon, termed "bronchial breathing," present. I strongly recommend you to take every opportunity of studying this sign, as it is a highly important one. You may get what is very like it by placing your stethoscope over the trachea, when you will hear a tubular, blowing kind of respiration, as of air passing to and fro through a hollow tube. "The *bronchial breathing* was very well marked indeed over the whole of the left lung posteriorly—best marked over the base, less intense over the next two-thirds, and but slight, and mixed with vesicular breathing, at the apex. The breathing over the left lung anteriorly was good and vesicular, as it was also over the whole of the right, except at the base, where some large, moist crepitation was audible." "There was increased *vocal resonance* where the bronchial breathing existed," there being that peculiar condition of voice termed "*bronchophony*," a condition which indicates that the voice, generated at the larynx, resounds more perfectly in the ear placed against the wall of the chest than it does in health, and seems as though it were actually formed in the corresponding bronchial tubes.

All these symptoms and physical signs led me to make the following diagnosis: "That the lower two-thirds of the posterior part of the left lung were hepatized, and that there was some condensation of the apex of the same lung; that the apex, though not yet hepatized, was quickly passing from the stage of *active congestion* into that of *red hepatization*." The signs upon which I chiefly relied in forming this diagnosis were, the bronchial breathing and the bronchophony, with the history of the disease and the general symptoms. And here I cannot too strongly warn you against the danger of paying exclusive attention to physical signs, and thereon founding your diagnosis, for by doing this you will very frequently fall into error. If I had attended to the physical signs alone in this case, I might have come to the conclusion that the disease consisted in *tubercular infiltration* of the lung, inasmuch as this condition will produce exactly the same signs as those to which the effusion of a fibrinous material into the air-cells, leading to the consolidation of the pulmonary tissue, gives rise, viz., dulness on percussion of a like character, bronchial breathing, and bronchophony. To decide whether the state of the lung was dependent on tubercular infiltration, or on the effusion of lymph into the air-cells, I took into consideration the history of the patient; and here the exposure to wet and cold, the shivering, the "stitch" in the side, and the absence of anything like the history of tubercles in himself or in his family, all led to the conclusion that the physical signs were produced by inflammation of the lung. Several cases of solidification of the lung have come before me, the result of inflammation, which had been pronounced to be phthisis, and the patients after a time got quite well. Let me put you on your guard against making such a mistake. In this case we derived no assistance from the characters of the sputa; for there was no expectoration, and this increased the difficulty of diagnosis.

Additional confirmation of our diagnosis in this case was derived from the fact that under treatment the signs of pneumonia began to disappear. On the day after his admission (26th) the pulse and respirations had greatly fallen, the former being 88, the latter 30 in a minute, the sound on percussion was much less dull, and distinct, large, moist crepitation could be heard below the spine of the scapula, though still lower down the breathing was bronchial, but mixed with some returning crepitation; all these signs indicating that the process of solution of the coagulable material in the air-cells and finer tubes had already commenced. On the 27th, evidence of the rapid resolution of the pneumonia existed; the pulse was 98, and the breathing 28, and the condition of the chest is thus recorded: "Posteriorly, over the apex of the left lung and left supra-spinal fossa, percussion is clear, indeed quite as clear as over the same regions on the right side; over the base of the left lung the percussion sound is less dull than yesterday, but still duller than that elicited over the corresponding point on the right side," showing that the lung was not then perfectly penetrated by air. "Below the spine of the left scapula, also, the sound on percussion is still slightly dull; in front, percussion is clear over both lungs. There is good, vesicular breathing in the upper two-thirds of the left lung behind," where the bronchial breathing had existed two days before, "and as one descends towards the base, slight, large, and moist crepitation becomes audible; while quite at the base distinct bronchial breathing is heard, with some crepitation on coughing and deep inspiration. The breathing is vesicular and puerile all down the right lung behind; in front the breathing in both lungs is good and vesicular."

The crepitation, which we find succeeding bronchial breathing, when accompanied with a diminution in the rate of the pulse and breathing, is one of the best signs of the resolution of pneumonia; but let me caution you against concluding that the crepitation *alone* is a favorable sign, inasmuch as it may be produced by the lung passing into the stage of *purulent infiltration.*

You must, therefore, endeavor by every means at your command to satisfy yourselves that this symptom, when present, does not depend on the lung advancing into a further state of degeneracy, which you may generally ascertain by carefully watching the rate of the pulse and respirations.

On the 28th, the patient was convalescent, and the appetite was returning; there was still some dulness over the lower part of the left lung posteriorly; with some bronchial breathing and moist crepitation quite at the base, showing that resolution was not yet perfect.

On the 29th (the 11th day of the disease), he was going on well, the pulse being 92, and the breathing 24, and the physical signs were good, the bronchial breathing having entirely disappeared. I have not seen him to-day, but have no doubt he is going on extremely well.

In my next lecture, gentlemen, I purpose speaking more at length of the condition of the urine in pneumonia, and of the treatment which it is advisable to adopt in this disease.

# LECTURE XII.

## ON PNEUMONIA.

GENTLEMEN,—I propose to resume to-day the consideration of the case of William Everitt, the little boy who has been suffering, as most of you are aware, from pneumonia. The diagnosis which we gave in the first instance was "that there was inflammation with consolidation of the inferior two-thirds of the left lung behind." That this was the condition of the lung we made out distinctly, and I explained to you in my last lecture the grounds upon which we came to this conclusion. Let me once more impress upon you the importance of bearing in mind the various points which enable one to say of a given case, not only that the existing symptoms are dependent on pneumonia, but also that they do *not* arise from any other morbid state of the lungs—in other words, that you should thoroughly understand what has been termed (although not very happily) *the differential diagnosis of pneumonia.*

The diseases from which you should be especially careful to distinguish pneumonia are the following:—

1st. Pleurisy, which, as you know, may be of two kinds, viz., *simple*, or *dry*, with or without an exudation of plastic lymph; and *pleurisy with effusion*, which leads pretty soon to the pouring out into the pleural cavity of a *fluid*, which may be either *serous* or *purulent.*

2d. That consolidation of lung which is due to the deposition of tubercles.

3d. That solidification of lung which is dependent on pulmonary apoplexy.

4th. A similar condition resulting from the deposition of cancerous matter in the lung structure.

All these conditions are apt to give rise to signs not very dissimilar from those produced by pneumonia.

In pleurisy, as in pneumonia, we have dulness on percussion over the diseased part; but in the former disease the dulness is more decided in its character than in the latter. In pleurisy, too, as in pneumonia, we have bronchial breathing and bronchophony; or more properly speaking, a peculiar modification of the voice which is termed *ægophony*, but which may easily be mistaken for bronchophony by an unpractised ear.

The presence of tubercles in the lungs gives rise to quickened respiration, dulness on percussion, bronchial breathing, and increased resonance of voice; and somewhat similar phenomena are likewise produced by *pulmonary apoplexy*, which term simply means an effusion of blood into the bronchial tubes and air-cells of a few or several, more or less contiguous, lobules. From some source of hemorrhage within the lung or opening into it, blood finds its way into one or more bronchial tubes, and during the act of inspiration is drawn down into the air-cells and finest bronchial ramifications, and thus a consolidation of lung, not unlike that which results from inflammation, is produced. The difference between the consolidation of lung dependent on pneumonia and that which is due to pulmonary apoplexy, is that in the one case the increased density is caused by the exudation of the plastic matter of the blood, with only a small portion of its coloring matter, into the air-cells and finer bronchial tubes, while in the other it results from the infiltration of the whole blood into the same channels.

In like manner, also, when cancerous matter is deposited in the lung (whether in the air-cells or in the areolar tissue around the lobules I am not prepared to say), dulness on percussion and other signs of consolidation are produced; and the dulness in this instance will always be found proportionate to the extent of the morbid growth.

When endeavoring to discriminate between pneumonia and pleurisy, you must keep in view that these two morbid conditions are very frequently associated, constituting what is termed *pleuro-pneumonia;* most cases of pneumonia, indeed, are of this last description, whilst *pleurisy*, *i. e.*, inflammation of the pleura, whether pulmonary or costal, but especially the latter, more frequently occurs as a separate and distinct affection.

If then a patient were admitted into the hospital with quickened breathing, pain in the side, the respiratory movements in one lung impeded, having previously had shivering, and having dulness on percussion over the lung in which the respiratory movements were embarrassed, how would you be able to satisfy yourselves that these symptoms depended on pleurisy and not on pneumonia? If the symptoms were due to *simple* pleurisy, the dulness would be but slight, and on putting the ear to the chest a *friction-sound* would be heard. The character of the pain would afford you some assistance: in pleurisy the pain is generally a sharp "stitch," while in pneumonia it is usually described as "dull." The reason of this is, that pleurisy often begins as a muscular affection, having its origin very frequently, I suspect, in rheumatic inflammation of the intercostal muscles, which spreads to the costal pleura. Upon the presence, then, of a friction-sound, upon the absence of marked dulness on percussion, and upon the absence, also, of any sign of crepitation, and upon the presence of vesicular breathing in the whole of the suspected lung, you can form a tolerably accurate diagnosis in favor of simple pleurisy and against pneumonia. But if rapidly followed, as pleurisy often is, by the effusion of a *small* quantity of a serous fluid into the pleural cavity, so that a *slight* layer of liquid intervenes between the pulmonary and costal pleuræ, the diagnosis between pleurisy and pneumonia is by no means so obvious; because, under such circumstances, a marked dulness on percussion is produced over a space corresponding to the extent of the effusion; but the dulness will be found on careful examination to be much more marked and decided than that which would be present in a case of simple pneumonia. In such a case, you would also have decided bronchial breathing; but, on testing the voice you would find bronchophony, not pure as in simple pneumonia, but in a modified form and exhibiting that peculiar tremulous, bleating character of the voice, whence it is called *ægophony*.

Again, how are you to determine in a given case that these signs depend, not on pneumonic solidification of the lung, but on pleurisy *with an abundant liquid* effusion? The proofs are these: if the symptoms depend on the presence of liquid in the cavity of the pleura, the dulness on percussion will be very complete and decided, and if the hand be placed flat against the thoracic parietes on the affected side and the patient made to speak, the vibrations of the voice will not be propagated to it.

If, on the other hand, the symptoms are due to pneumonia, vocal vibrations will be more or less freely communicated to the hand placed against the wall of the chest, the freedom of propagation being influenced mainly by the permeability of the bronchial passages to air.

When these two signs—marked dulness on percussion, and absence of vocal vibrations—exist together, the evidence which they afford is conclusive as regards the presence of a liquid effusion in the cavity of the pleura. In such a case, too, bronchophony is replaced by ægophony, unless the effusion be very large, and the bronchial breathing has not the same intense character which it would have in the case of a lung consolidated by pneumonia, nor does it convey to the same extent the idea of nearness.

There are two points which I must here impress upon you. The first is, that now and then you meet with a case in which there has been a former attack of pleurisy which has left adhesions. In such a case, you may have vocal vibrations present, yet there may be marked dulness and ægophony. The adhesions are sufficient to propagate the vocal vibrations from the lung to the wall of the chest.

The second point is, that sometimes the voice is feeble, and its vibrations cannot be propagated beyond a certain portion of the bronchial tree, and not at all to the thoracic parietes. This occurs chiefly in women; sometimes in weakly men.

In addition to all these phenomena, there is, generally speaking, something in the history of the case which affords aid in arriving at an accurate diagnosis: thus, pleurisy is more frequently connected with a rheumatic state of the constitution, or *diathesis*, than pneumonia; and in its early stages the symptoms of the former disease are much more severe as regards pain, but much less urgent as regards the affection of the breathing, than those of the latter. There is generally less heat of skin in pleurisy than in pneumonia; and the characteristics of pleurisy, on the whole, approximate those of a (so-called) *sthenic* disease, while the characters of pneumonia are more nearly allied to those of an *asthenic* affection; and patients laboring under inflammation of the pleura bear bleeding better than those who are suffering from inflammation of the lung.

With regard to the differential diagnosis of pneumonia and pulmonary apoplexy, cancer, and tubercle, you must be guided in a great degree by the history of the case. With respect to tubercular deposits, it is important to note the *position* of the dulness on percussion; in tubercular disease this occurs at the apices of the lungs far more frequently than anywhere else; while in pneumonia it is usually found over the base of these organs. The nature of the *expectoration* also affords valuable information; thus, if tubercles be present, but in a crude state, they may give rise to little or no expectoration; or the fluid spat up may be colorless, and exhibit very much the appearance of saliva; or it may be a glairy mucus; or, as the tubercles soften it may assume more or less the characters of pus; or at a former time or times blood in small or large quantity may have been spat up; but in genuine cases of pneumonia the sputum in the early stages is almost invariably rust-colored, viscid, and tenacious. Do not, however, lose sight of the fact that in pneumonia of the apex there is very often a total absence of expectoration.

In cancerous disease of the lungs, the history of the case, as I just now observed, comes to our aid; and here, too, there is generally an absence of fever, and the dulness on percussion is usually less extensive than in an ordinary case of pneumonia, inasmuch as the cancerous matter, in consequence of its deposition for the most part in detached masses, does not usually consolidate a large portion of the lung. In such a case, also, there will be absence of the breath-sounds to a greater or less extent, and, associated with this, a material deficiency in the ordinary movements of the thoracic walls will generally be found. In addition to all these, there is the cancerous *cachexia*, as it is called, and that peculiar, enfeebled, more or less anæmic, habit of system, which an experienced eye readily detects; while the family history of the patient sometimes affords important information as to the nature of the malady. I need scarcely add that this remark applies even more strongly to tubercular disease.

In the case of pulmonary apoplexy, we receive considerable assistance from the fact of the occurrence of a pretty copious hemorrhage, by which this state of lung is invariably produced. This morbid condition is most commonly associated with disease of the heart, and is generally caused by something which either interferes with the return of the blood to the left side of the heart, or prevents its free flow through the pulmonary bloodvessels, the consequence of the obstruction being the rupture of some of them, the effusion of blood, and the consolidation of those parts of the lung into which this blood obtains entrance.

Referring once more to the diagnosis between pneumonic and tubercular consolidation of the lung, let me put you on your guard against being led astray by a peculiar form of chronic pneumonia, which I generally call strumous pneumonia. It occurs in children and young persons of strumous constitution, and almost always attacks the apex of one lung; generally there is no expectoration, and if it occur it is not colored. The lung becomes gradually solid, and as gradually undergoes resolution. The progress of the case affords the best aid to its diagnosis, and a practitioner who is cautious in forming his opinions, and on the lookout for such an occurrence, will seldom go astray.

Having made these introductory remarks, let me now proceed to the consideration of the condition of the urine in pneumonia; and I have here to express my thanks to my friend, Dr. Evans, as far as regards this secretion, for the able manner in which he has worked out the case, thus enabling me to lay before you the constitution of the urine of this patient, on almost every day after his admission into the hospital until his restoration to health.

The composition of the urine in pneumonia is a matter of recent observation. I am anxious to draw your attention to it, because the notes of cases of this disease are incomplete and unsatisfactory in a clinical point of view, unless the condition of the urine from day to day is carefully recorded. There are certain salts constantly existing in normal urine, which become totally, or almost totally, deficient in this secretion, at the period when hepatization of the lung is complete, and are not fully restored until resolution is established.

To make this subject more intelligible, let me call your attention to some of the leading points connected with the composition of the urine in the state of health.

The urine consists of a large proportion of water which holds in solution certain solid matters. These solid ingredients are of two kinds, viz., *organic* and *saline;* the former containing *urea, uric acid*, and certain mystical chemical compounds termed *ex-*

*tractives* or *extractive matters*, which are further distinguished as *water extractive* and *alcohol extractive*, of the exact nature of which we shall some day know something more; the latter comprising the *sulphates of potash and soda*, *alkaline* and *earthy phosphates*, and *chloride of sodium*, with occasionally a greater or less quantity of *chloride of ammonium*, or, as it is generally termed, *hydrochlorate*, or *muriate of ammonia.*

Now, in the urine in pneumonia, there is a deficiency in all the *saline* ingredients, but the salt whose diminution is most marked is *chloride of sodium* (common salt). This point was first made out by Redtenbacher in Germany, who, upon observations on eighty cases of this disease, clearly established the fact, that during the whole period of hepatization, and, indeed, until resolution is freely established, chloride of sodium is wholly, or almost wholly, absent from the urine.

The presence or abscence of this *chloride* in the urine may be ascertained by a chemical test, so simple in its nature, and so readily used, that it is in the power of every one to search for this substance as easily as for albumen. The chemical operation to which I allude is as follows: about a drachm of the urine should be placed in a test-tube and acidulated with a little nitric acid; to this a few drops of a solution of nitrate of silver should now be added, when, if any chloride be present, a white flocculent precipitate (*chloride of silver*) will occur; and the *quantity* of chloride in the urine may be roughly estimated by noting the bulk which this precipitate occupies, after having been allowed to subside to the bottom of the test-tube for a short time, though, of course, when accurate results are sought for, an altogether different mode of proceeding must be adopted. If, on the other hand, no precipitate takes place on the addition of the solution of nitrate of silver, you may conclude positively and certainly that no chloride is present in the urine. This process is not always perfectly certain, inasmuch as it is invalidated by the occasional presence of chloride of ammonium in the urine, which, like chloride of sodium, gives a similar white flocculent precipitate when treated in this way; but as the presence of chloride of ammonium is a rare occurrence, this test in the large majority of instances answers sufficiently well for ordinary purposes, and the evidence which it affords, when there is a total absence of any precipitate, is quite conclusive as to the entire disappearance of chloride of sodium from the urine.

I will now relate to you what happened with respect to this matter in the case we are considering.

The lad, you will bear in mind, was taken ill on October 19th—on that day he first shivered—on the 21st he was admitted into the hospital; and the diagnosis which was then made, was, as I told you in my last lecture, that the lower two-thirds of the posterior part of the left lung were in a state of hepatization, and there was some condensation of the apex of the same lung. He was put under treatment on the 21st, and on that day his urine was tested with nitric acid and nitrate of silver in the way which I just now described. To our great surprise a copious white precipitate was produced, affording a *primâ facie* indication that chloride of sodium was present, despite of the pneumonic consolidation. This was, however, clearly exceptional, from some cause which we could not ascertain, or it was due to the temporary presence of chloride of ammonium. Upon this point we could gather no precise information, as, unfortunately, no more urine could be obtained on this day to subject to a more exact method of analysis. On the following day, however, which, reckoning from the occurrence of the shivering, was the *eighth* of the disease, a sufficient quantity of urine was obtained to submit to minute analysis, so that we did not depend on the simple mode of testing to which I have already referred. On this day, while a great portion of the left lung was in a state of simple hepatization, with, perhaps, a little resolution just commencing, scarcely a trace of chloride of sodium existed in the urine; so that it was a reasonable inference that on the preceding day the precipitate, which was produced on the addition of nitric acid and nitrate of silver, was due to the presence of chloride of ammonium; and an analysis of 100 grains of the *solid matters* of the urine on this day gave *organic matters* 97·034 and *fixed salts* 2·966. Now the relative proportion of the organic to the saline constituents of the solid matters of ordinary healthy urine is as from 70 to 75 are to from 30 to 25; or, in round numbers, the composition of 100 grains of the solid matters of healthy urine may be represented as *organic matters*=75, *fixed salts*=25. You see, then, what a remarkable diminution had taken place in the amount of the saline constituents of the urine of this patient on the eighth day of the pneumonia—from 25 to barely 3 parts in a hundred!

On the ninth day the physical signs indicated that a general resolution of the inflammation had taken place; and an analysis of the urine showed that this fluid contained merely the slightest trace of chloride of sodium, while 100 grains of its *solid matters* were composed of 97·938 of *organic constituents*, and 2·062 of *fixed salts*—even less than the preceding day.

On the tenth day convalescence had considerably advanced; the chloride of sodium was, as yet, *entirely* absent from the urine, but the proportion between the *saline* and *organic* constituents had somewhat altered, the former having slightly increased, the numbers being, *organic matters*=97·702, *fixed salts*=2·298.

The urine of the twelfth and that of the thirteenth days of the disease were unfortunately mixed together, so that an analysis of each separately was, of course, impossible. By this time the patient had completely thrown off all traces of the pneumonia; air freely permeated every part of the lung, consolidation had disappeared, and he was up and about, and hungry, but still on the same diet (and this is a very important point) upon which he was first put when brought into the hospital, viz., the hospital *milk diet* and *beef tea*. We now found that a remarkable change in the relative proportion of the organic to the saline constituents of the urine had suddenly taken place; the organic matters, instead of being 97 per cent. of the solids, had fallen to 72·814, while the fixed salts had risen from rather more than 2 per cent. up to 27·186; showing how very rapidly this change must have occurred, the patient having still the same diet as on the five preceding days, so as completely to exclude the idea of its being due to the mode of feeding. On this same day the chloride of sodium had returned to the urine, but in rather less than its normal amount, viz., 4·321 in 1000 grains of urine.

On the fourteenth and fifteenth days analyses of the urine gave similar results, and chloride of sodium was present on the fourteenth, in rather less than its normal proportion; but on the fifteenth, the patient having been put on *middle diet* the previous day, it had increased from 4 to 7·180 in 1000 grains of urine.

All these are very curious facts if taken alone, as carefully observed in this one case; but if you refer to the thirtieth volume of *Medico-Chirurgical Transactions*, you will find a very interesting paper by Dr. Beale, in which the same points are fully established as founded on observations which were made on several cases. The question which these facts naturally suggest, and a most important one it is for pathology, is, "What becomes of the chloride of sodium, under these circumstances, if it does not pass out in the urine?" The object of carrying off these saline substances in the urine is probably to convey away certain effete matters derived from the waste of the tissues, or else introduced with the food; but during the first five days this patient was under treatment, he was daily taking a certain amount of chloride of sodium in his food (*milk diet and beef tea*), which did not find its way out of the body in the urine. Dr. Beale has shown that a large quantity of chloride of sodium under these circumstances accumulates in the *sputa* and in the *inflamed portion of the lung*, while the proportion of this salt in the *serum of the blood* falls below its normal standard. From this it would appear that there exists in pneumonia some attraction between the inflamed lung and chloride of sodium; for if this substance be in excess in the inflamed lung, while at the same time it is deficient in the blood and urine, the inference is immediately suggested that, in some way or other, it becomes attracted to the inflamed pulmonary tissue. Upon the precise object of this process it is difficult to speculate: but we must, for the present at least, take it as a fact, and endeavor to ascertain whether a similar state of things occurs in acute inflammations of other organs, or in bronchitis, &c. Any of you, who have ever had a severe attack of coryza, must have noticed that the nasal secretions have a very saltish taste, as if, in this affection, chloride of sodium were attracted in undue quantity to the inflamed surface.

Another question which suggests itself here is—Why does the chloride of sodium return so quickly into the circulation and urine? For no sooner does the attracting force set up by the inflamed lung cease to operate, than the chloride finds its way out of the system through the ordinary channel.

I may here again allude to a point which I before mentioned, viz., that a diminution takes place in all the other fixed salts of the urine as well as in the chloride of sodium. The latter, it is true, disappears altogether; but the sulphates and phosphates become remarkably diminished; for, as I just now told you, analytical examinations of the urine of this patient on the

| | Anal. I.<br>Eighth Day of Disease.<br>Sp. Gr. 1013. | | | Anal. II.<br>Ninth Day of Disease.<br>Sp. Gr. 1013. | | | Anal. III.<br>Tenth Day of Disease.<br>Sp. Gr. 1012. | | | Anal. IV.<br>Twelfth and Thirteenth Day of Disease.<br>Sp. Gr. 1010. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | In 1000 Grains of Urine. | In 100 Grains of Solid Matter. | In 100 Grains of Fixed Salts. | In 1000 Grains of Urine. | In 100 Grains of Solid Matter. | In 100 Grains of Fixed Salts. | In 1000 Grains of Urine. | In 100 Grains of Solid Matter. | In 100 Grains of Fixed Salts. | In 1000 Grains of Urine. | In 100 Grains of Solid Matter. | In 100 Grains of Fixed Salts. |
| Water, . . . . | 944·277 | | | 957 875 | | | 969·028 | | | 972·825 | | |
| Solid Matter, . . | 55·723 | | | 42·125 | | | 30·972 | | | 27·175 | | |
| Organic Matter, . | 54·070 | 97·034 | | 41·256 | 97·938 | | 30 260 | 97·702 | | 19·787 | 72·814 | |
| Fixed Salts, . . . | 1·653 | 2·966 | | ·869 | 2·062 | | ·712 | 2·298 | | 7·388 | 27·186 | |
| Urea, Extractive, & Ammon. Salts, | 53·302 | 95·650 | | 38·927 | 92·410 | | 27·428 | 88·559 | | 18·733 | 68·936 | |
| Uric Acid, . . . | ·768 | 1 384 | | 2·329 | 5·528 | | 2·832 | 9·143 | | 1·054 | 3·878 | |
| Chloride of Sodium, | A slight trace. | A trace. | A trace. | A slight trace. | A trace. | A trace. | Not a trace. | None. | None. | 4·321 | 15·900 | 58·486 |
| Sulphuric Acid, . | ·662 | 1·186 | 40·048 | ·208 | ·493 | 23·935 | ·308 | ·994 | 43·258 | 1·313 | 4·831 | 17·772 |
| Phosphoric Acid, . | ·512 | ·918 | 30·973 | ·244 | ·579 | 28·078 | ·110 | ·355 | 15·449 | 1·115 | 4·103 | 15·092 |

| | Anal. V. Fourteenth Day of Disease. Sp. Gr. 1009. | | | Anal. VI. Fifteenth Day of Disease. Sp. Gr. 1012. | | | Anal. VII. Seventeenth Day of Disease. Sp. Gr. 1020. | | | Anal. VIII. Twenty-first Day of Disease. Sp. Gr. 1023. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | In 1000 Grains of Urine. | In 100 Grains of Solid Matter. | In 100 Grains of Fixed Salts. | In 1000 Grains of Urine. | In 100 Grains of Solid Matter. | In 100 Grains of Fixed Salts. | In 1000 Grains of Urine. | In 100 Grains of Solid Matter. | In 100 Grains of Fixed Salts. | In 1000 Grains of Urine. | In 100 Grains of Solid Matter. | In 100 Grains of Fixed Salts. |
| Water, . . . . | 978·942 | | | 964·617 | | | 950·694 | | | 943·528 | | |
| Solid Matter, . . | 21·058 | | | 35·383 | | | 49·306 | | | 56·472 | | |
| Organic Matter, . | 13·444 | 63·843 | | 24·408 | 68·983 | | 38·640 | 78·368 | | 43·462 | 76·962 | |
| Fixed Salts, . . . | 7·614 | 36·157 | | 10·975 | 31·017 | | 10·666 | 21·632 | | 13·010 | 23·038 | |
| Urea, Extractive, & Ammon. Salts, | 12·851 | 61·027 | | 23·379 | 66·075 | | 37·397 | 75·848 | | 42·810 | 75·808 | |
| Uric Acid, . . . | ·593 | 2·816 | | 1·029 | 2·908 | | 1·243 | 2·520 | | ·652 | 1·154 | |
| Chloride of Sodium, | 4·877 | 23·159 | 64·053 | 7·180 | 20·292 | 65·330 | 3·429 | 6·954 | 32·148 | 5·995 | 10·615 | 46·079 |
| Sulphuric Acid, . | ·757 | 3·594 | 9·942 | 1·381 | 3·903 | 12·583 | 1·774 | 3·780 | 16·632 | 1·399 | 2·477 | 10·753 |
| Phosphoric Acid, . | ·522 | 2·748 | 6·855 | 1·487 | 4.202 | 13·548 | 1·771 | 3·774 | 16·604 | 2·891 | 5·101 | 22·221 |

eighth, ninth, and tenth days of the disease, gave 2·966, 2·062, and 2·298 respectively, as the amount of fixed saline matter in 100 grains of the dried solids of the urine, instead of the normal proportion of 25 per cent.

To enable you more clearly to comprehend these curious and interesting facts with reference to the urine, which I have just mentioned, I have here a table drawn up by Dr. Conway Evans, showing the composition of the urine of this patient on the eighth, ninth, tenth, twelfth, and thirteenth, fourteenth, fifteenth, seventeenth, and twenty-first days of the disease. By carefully studying these analyses you will notice several other interesting points besides those to which I have alluded, and which I regret time does not permit me to enter into. With respect to the table, let me add, that the first column shows the composition of 1000 grains of urine; the second column shows the composition of 100 grains of the solid constituents of the urine; or, in other words, the percentage of the various components of the solid ingredients; while the third column represents the amount of chloride of sodium, and of sulphuric and phosphoric acids, contained in 100 grains of the fixed saline constituents.

One conclusion may certainly be inferred from these curious chemical details, namely, that the disease of which we are speaking involves profound changes in the chemistry of life—in the interchanges between the blood and the tissues, and in the chemical constitution of the blood itself. And not only so, but the process of *recovery* from the disease involves analogous chemical changes in an opposite direction, showing that there is in the human system a wonderful power of restoring the injuries inflicted by disease. When we shall have obtained a clearer insight into the recondite processes by which repair is effected in the animal body, we shall be in a better position to assign to our experimental interferences their proper position either as helps or hindrances to this inherent *vis medicatrix.*

Let me, now, call your attention to the treatment which was pursued in this case. The lad first applied at the hospital on October 21st, and he continued as an out-patient until the 25th; and during this period he was treated with quarter-grain doses of tartar emetic administered every four hours, which did not prevent the progress to hepatization. On the evening of his admission (October 25th) his pulse was 120, and his respirations were 48, in a minute. The treatment adopted consisted in the exhibition of three-drachm doses of the solution of acetate of ammonia every two hours, with two drachms of wine every four hours, a moderate supply of beef tea, milk diet, and the application of turpentine stupes over the left side of the chest.

On the following day, the pulse was 100, and the respirations were 36; on the 27th, the pulse was 76, and the breathing 28 (the numbers throughout are those of the evening), and general resolution of the inflammation had taken place; air was admitted freely into the lung, and the dulness had become much less both in extent and intensity. From this day the improvement was very rapid; the pulse very quickly returned to its normal condition; and on the fourth day of the treatment (the tenth from the occurrence of the shivering), the patient was pronounced convalescent. On November 1st, the diet was changed from one consisting of milk, bread, and beef tea, to animal and vegetable food in the ordinary way.

The object of the treatment pursued in this case, and which, with slight modification, I adopt in all others, is to imitate as far as possible the course which nature pursues in this disease. If you watch the changes which a patient in pneumonia undergoes in his progress towards convalescence, you will find that about the eighth, ninth, or tenth day of the disease, or from the tenth to the twelfth day, more or less sweating almost always takes place. Sometimes the sweating occurs freely, and it is then considered *critical.* Sometimes, too, a *critical* discharge of purulent matter from the lung takes place by expectoration; now and then the kidneys about this time secrete much more urine than previously; and occasionally, though certainly very rarely, a *critical* diarrhœa occurs. Always chloride of sodium begins to escape from the system through the urine, from which, during the height of the disease, it had been excluded. All this looks as if a *something,* which caused the morbid change in the lung, were eliminated from the system in one or more of the secretions; and, therefore, to promote these should be the aim of our treatment.

It may be laid down, I think, as a general rule that large evacuations by sweating may be employed more freely and with less disadvantage to patients than by any other secretion; and after a patient with pneumonia has perspired freely, there almost always occurs a marked change for the better. In some diseases profuse sweating takes place at an early period, as, for example, in acute rheumatism and gout; in such cases it is evidently set up for conservative purposes, and to check it is highly unwise, unless it seems to be running the patient down.

A large number of cases of pneumonia are of rheumatic origin, and occur, as I told you in my last lecture, in rheumatic subjects. Indeed, in rheumatic fever many cases of pneumonia begin, while some cases of inflammation of the lung pass on and end in an attack of acute rheumatism. And another reason why you should especially promote the sweating process in such instances is because in rheumatic fever and in gout the morbid matters, whatever they be, appear, in great measure at least, to be carried off by the skin.

Bear in mind, as a reason against adopting a violent course of treatment, that pneumonia has, on the whole, a decided tendency to get well of itself. I have never had the courage, and I don't think I ever shall, to let a patient with pneumonia alone, and trust to the *expectant system*, as it is called; and I am not acquainted with any satisfactory observations which have been made on this point; but there can be little doubt, I think, that the general tendency of the disease is towards recovery.

From these reasons, then, you will readily see that there is no necessity for having recourse to violent antiphlogistic (so called) measures in cases of pneumonia; moreover, I tell you that in the course of more than thirty years' experience I have tried all ways—calomel and opium—bleeding—tartar emetic—and the various others which have been proposed—and if I had found that under any one of these plans no death, or even only a very few deaths, had occurred, to that mode of treatment I should have steadfastly adhered; but I found that under each one of these a great number of deaths occurred, and that it frequently happened to me to be called in to patients who had been bled several times, and yet to find the lung in a decided state of hepatization, although the bleeding measures had been adopted at the earliest moment. In the very case before us the patient had been taking tartar emetic four days, and during this time the hepatization had steadily increased; but as soon as this mode of treatment was altered for a milder plan, the depressing influence of the tartar emetic removed, and sweating promoted, resolution of the inflammation took place with great rapidity.

Among all the cases of pneumonia which I have treated with tartar emetic, I found that none did so well as those in which a toleration of the medicine was established *early;* where it got into the system, but produced no sensible effect, or merely sweating. But the cases in which this remedy caused vomiting, and, more especially, those in which it purged, by no means turned out so satisfactorily as those in which it acted as a diaphoretic only.

What has astonished me in most of the instances of this disease that I have treated or seen treated in the manner which I now recommend to your notice, is the great rapidity with which all traces of the affection are thrown off, and the shortness of the period which elapses before the patient is restored to his usual state of health.

# LECTURE XIII.

## *On Pneumonia.*

Let me to-day, Gentlemen, conclude the observations which I made in two former Lectures this session on the subject of pneumonia, by calling your attention to two cases of this disease recently in the hospital. One of these having proved fatal, I avail myself of the opportunity of bringing before you, in a more special manner than I have hitherto been able to do, the circumstances which tend to promote a fatal or a favorable termination in any given case of this disease.

You know that I have often insisted upon the importance of paying special attention to fatal cases. Why does any man die of a curable disease? As long as this question can be asked, the *Ars Medica* is in a doubtful position; and it is far better to look candidly and honestly into all the particulars which belong to a fatal case, than to flatter ourselves with the more agreeable contemplation of those which have recovered.

In all fatal cases you should review your practice, with the object of endeavoring to ascertain whether the treatment which has been adopted has had any share in contributing to the fatal result, whether there has been any neglect on the part of the attendants, or whether, from some peculiarity of constitution, or from the age of the patient, or some other circumstance, certain conditions have existed which have tended more to an unfavorable than to a favorable issue. All these investigations are of great practical utility, inasmuch as they enable the medical man to form a sober judgment of the plan of treatment which he has pursued, and at the same time, if properly conducted, they afford him a greater insight into the nature of the disease, and enable him to speak more decidedly in any subsequent case as to the final issue. And when you get into practice for yourselves, you will find that there is nothing more important than that you should be able to determine satisfactorily whether your *prognosis* shall be favorable or unfavorable, as you will always find your patient surrounded by anxious friends, who look to the doctor almost as if in his hands "lay the issues of life and death."

With these prefatory remarks, then, let me proceed at once to the consideration of the fatal case of pneumonia which has occurred so lately under my care in the hospital.

CASE LXXXII. (Vol. xli, p. 165.) The patient was a man named Charles Johnson, æt. 48, who was admitted into Fisk ward on January 16th, 1854; and the date of the disease was from January 12th, *i. e.*, four days before he entered the hospital. I do not propose going into any lengthened detail of the symptoms in this case, feeling confident that from all I have told you in my former lectures on this subject, you must all be quite *au fait* with respect to the signs of pneumonia. But let me remark, that in keeping notes of cases of this affection, you should endeavor to ascertain as accurately as possible the date of the commencement of the complaint, for this point is of great importance with reference to the clinical history of pneumonia. What symptom, you will ask me, indicates the commencement of the disease? This question may be answered in a word—*rigors*. They do not always occur distinctly, and sometimes their occurrence is not noticed by the patient, but when they can be fixed to a date, they form the starting-point for the disease. This man had distinct rigors on January 12th, and we consequently take this day as the starting-point of the pneumonia. He belonged to a profession which is not perhaps the most conducive to health, for "he gained his livelihood by tumbling about the streets." Though not distinctly intemperate, according to his own account, his habits were, no doubt, not of the most regular kind; and "in consequence of the peculiar nature of his calling, he was continually exposed to wet and cold," conditions exceedingly liable to induce affections of the chest. "His illness commenced," as I just now said, "on January 12th, when, after having his feet wet for some hours, he was seized with rigors and a feeling of uneasiness in the chest. This was so severe as to prevent him from following his usual occupation, and on the 16th he began to suffer from pain in the left side of the chest, and shortness of breath. These symptoms increasing in severity, on the evening of that day he was brought into the hospital, when his pulse was 126, and his breathing 32 in a minute, his skin hot, his tongue furred, and he complained of pain in the left side of the chest." In short, he had all the symptoms of pleuro-pneumonia of the left side, there being bronchial breathing and bronchophony, marked dulness on percussion, and absence of vocal vibrations, showing the existence of a slight pleuritic effusion.

He was immediately put under the plan of treatment which you know I have of late been accustomed to employ in all cases of pneumonia, viz., that which has a tendency to promote sweating, and which simply aims at imitating Nature in the course which she adopts in getting rid of the disease; for it appears to be by an augmentation in the secretions of the skin and of other organs, that the morbid materials, which, by their accumulation in the system, seem to irritate the lung and to produce, or at all events to keep up, the disease, are eliminated in the most beneficial way.

An important feature in this plan of treatment is to counteract as far as possible every influence of a depressing nature; and *though the exhibition of stimulants does not form a necessary part of it*, still the aim should be to uphold the patient's strength. This is best done by providing for him frequent supplies of good beef tea or other animal broth. Solid food you cannot give in these cases, for not only is it digested with great difficulty, but also the want of appetite prevents it being taken; but by small quantities of beef tea frequently supplied, a large amount of easily appropriated nutriment may be administered in the course of twenty-four hours. When the vital powers are clearly depressed, with a pulse *increasing in quickness*, or when the patient has been previously accustomed to live well, or in the habit of indulging in the use of alcoholic drinks, then wine or brandy, or, what is really the proper way of expressing it, *alcoholic food* must be exhibited. Our patient was in a depressed state on his admission, and there were also strong suspicions of his having been to a certain extent intemperate in his habits, and on these accounts he was put, at once, upon half an ounce of wine every four hours. In addition to this, he was ordered six drachms of the *liquor ammoniæ acetatis* every three hours, turpentine stupes to the chest, with a free supply of beef tea and milk. The diagnosis which was formed was this: "there is a great patch of pneumonia at the lower part of the side and front of the left lung, having a tendency to spread, with, probably, some degree of pleurisy."

On the sixth and seventh days of the disease—and I may here remark that in keeping notes of the cases of pneumonia, it is exceedingly convenient for clinical purposes to note not only the day of the month, but also *the day of the disease*, a point which both my clinical clerks, I am very glad to find, have most properly attended to—on the sixth and seventh days of the pneumonia there was a marked improvement both in the pulse and respiration, on the sixth day the pulse being 104 and the respirations 32, and on the seventh the numbers being 100 and 40, while on the fourth day they were 126 and 32, and on the fifth 112 and 40 respectively. All this looked well, and I was beginning to entertain hopes that the patient would recover, although the case was undoubtedly a very severe one. On the eighth day, however, matters seemed to take a very unfavorable course, and I wish to call your attention to the circumstances under which this change took place. The report says, "he slept better last night, but he does not look nearly so well to-day; his face is dusky and anxious, and he lies with his mouth and eyes half open; he has great difficulty in expelling the viscid sputa from his mouth; pulse 116, very feeble; respirations 30." There were now, also, signs that the disease, which at first was situated in the lower third of the left lung, was rather rapidly spreading in the upward direction; and in consequence of the feeble character of the pulse he was ordered half an ounce of brandy every hour, with a draught every third hour containing five grains of sesquicarbonate of ammonia with half a drachm of chloric ether. Now the point to which I wish particularly to direct your attention here is, that the unfavorable change immediately succeeded a good night's sleep; and I have no doubt that it arose out of that lengthened sleep. I have frequently seen cases in which a long-continued sleep, occurring when the system was much depressed from any cause, has left the patient much worse; and for this reason, that the continued sleep deprives the patient of the due amount of nourishment and support which should be supplied him. This often happens in typhus fever; the patient sleeps five or six hours continuously, and his friends or the nurse imagine that the best thing that can be done is to allow him to sleep on: at the end of this time he wakes up very much exhausted, and with his pulse weaker and more rapid than before he went to sleep. As a general rule, you should not, in my judgment, allow patients in a depressed state of the system to sleep longer than two or three hours at a time, often not longer than an hour, but they should be awakened gently at specified times and proper nourishment supplied them. Nor will this proceeding prevent them from going to sleep again; on the contrary, patients so treated, generally speaking, fall off to sleep immediately after they have taken their nourishment, and the sleep so obtained is sound and refreshing.

I remember (though no account of the circumstance is recorded in the notes of the case) that there was good reason for believing that this patient was neglected one night by the night-nurse, and, if my memory does not deceive me, this occurrence took place on the night of the eighth day of the disease. I should also tell you that on the sixth and seventh days, although a general improvement took place, the patient suffered much from diarrhœa, which we were obliged to check by astringents; but this had entirely ceased on the eighth day. From the ninth day the patient declined; on this day the pulse was 120, and respirations were 34 a minute, and the physical signs indicated a decided increase of the pneumonia, the whole of the left lung appearing to be involved in the disease. On the tenth day the patient was reported to be sinking, and on the morning of the eleventh he died.

In this case observations were made on the composition of the urine and sputa, and it was found that the former contained a very small, but decided, quantity of albumen, with which certain granular *casts* of the uriniferous tubes indicated incipient chronic disease of the kidneys; and this circumstance, of course, tended to render the prognosis still more unfavorable. On the fifth day of the disease the urine contained only a slight trace of fixed chloride, and on the sixth day the report was the same; but on the seventh day this excretion contained neither chlorides nor sulphates, but in 1000 grains of urine there were nearly four grains (3·962) of phosphoric acid; while in 100 grains of the solid matters of the urine there were 7·72 of this acid, and in 100 grains of the fixed salts 60·757. This excessive amount of phosphates in the urine of this day is a very curious feature in the case, and probably increased the nervous depression.

The observations of Redtenbacher with respect to chloride of sodium in this disease were confined to the urine; but the investigations of Dr. Beale show that, while this salt vanishes from this fluid, it makes its appearance in considerable quantity in the sputa. In this case, on the sixth day of the disease, 100 parts of the solid matters of the sputa contained 8·44 of chloride of sodium; while 100 grains of the fixed salts contained 48·243 of this substance. On the seventh day 100 grains of the solids of the sputa contained 14·544 of chloride of sodium, while 100 grains of the fixed salts contained 58·824; and on the eighth day, in 100 parts of the solid matters of the sputa, there were 10·929 of this salt, while in 100 parts of the fixed salts there were 67·35.

By carefully reviewing these tabulated results of the analyses of the sputa and urine, which were made by my friend, Dr. Evans, you will readily understand the interesting points in the composition of these fluids which I have just mentioned. The analyses of the *sputa* are those of the sixth, seventh and eighth days of the disease; those of the *urine* are of the seventh and eighth days. The first column of each table represents the composition of 1000 grains of *sputum* or *urine* respectively; the second column exhibits the composition of 100 grains of the *solid matters* of either; while the third column shows the proportion of chloride of sodium, sulphuric acid, and phosphoric acid entering into the composition of 100 grains of the *fixed salts* in each.

I very much wish that some of you would take up this subject of the exact chemistry of the *excreta* in the various stages of pneumonia, both towards a destructive as well as a reparative result. It will be only by such researches that we shall arrive at more certain views of the pathology of this disease, and through it of other acute parenchymatous inflammations.

In one or two cases of pneumonia the sputa have been found to contain grape-sugar in greater or less quantity, and this fact is highly interesting both as bearing on the pathology of the disease, and also, in a general way, on the office of the respiratory organs in consuming the saccharine matter which is formed in the system. But in this case, though the sputa of the sixth,

| | Anal. I. Sputum. Sixth Day of Disease. | | | Anal. II. Sputum. Seventh Day of Disease. | | | Anal. III. Sputum. Eighth Day of Disease. | | |
|---|---|---|---|---|---|---|---|---|---|
| | In 1000 Grains of Sputum. | In 100 Grains of Solid Matters. | In 100 Grains of Fixed Salts. | In 1000 Grains of Sputum. | In 100 Grains of Solid Matters. | In 100 Grains of Fixed Salts. | In 1000 Grains of Sputum. | In 100 Grains of Solid Matters. | In 100 Grains of Fixed Salts. |
| Water, . . . . . . . | 961·923 | | | 952·676 | | | 950·739 | | |
| Solid Matter, . . . . | 38·077 | | | 47·324 | | | 49·261 | | |
| Organic Matter, . . . | 31·415 | 82·504 | | 35·623 | 75·274 | | 41·267 | 83·772 | |
| Fixed Salts, . . . . | 6·662 | 17·496 | | 11·701 | 24·726 | | 7·994 | 16·228 | |
| Chloride of Sodium, . . | 3·214 | 8·440 | 48·243 | 6·883 | 14·544 | 58·824 | 5·384 | 10·929 | 67·350 |
| Sulphuric Acid, . . . | 1·739 | 4·567 | 26·103 | 1·907 | 4·029 | 16·297 | ·769 | 1·561 | 9·620 |
| Phosphoric Acid, . . . | ·972 | 2·552 | 14·590 | ·956 | 2·020 | 8·170 | ·509 | 1·033 | 6·367 |

| | ANAL. IV.<br>Urine.<br>Seventh Day of Disease.<br>Sp. Gr. 1017·2. | | |
|---|---|---|---|
| | In 1000 Grains of Urine. | In 100 Grains of Solid Matter. | In 100 Grains of Fixed Salts. |
| Water, . . . . . . . . . . . | 948·683 | | |
| Solid matter, . . . . . . . . . | 51·317 | | |
| Organic Matter, . . . . . . . . | 44·796 | 87·293 | |
| Fixed Salts, . . . . . . . . . | 6·521 | 12·707 | |
| Uric Acid, . . . . . . . . . . | 1·130 | 2·201 | |
| Urea, Albumen, Extractive, and Ammoniacal Salts, . . . . . | 43·666 | 85·092 | |
| Chloride of Sodium, . . . . . . . | None. | None. | None. |
| Sulphuric Acid, . . . . . . . . | None. | None. | None. |
| Phosphoric Acid, . . . . . . . . | 3·962 | 7·720 | 60·757 |

| | ANAL. V.<br>Urine.<br>Eighth Day of Disease.<br>Sp. Gr. 1012·6. | | |
|---|---|---|---|
| | In 1000 Grains of Urine. | In 100 Grains of Solid Matter. | In 100 Grains of Fixed Salts. |
| Water, . . . . . . . . . . . | 964·166 | | |
| Solid Matter, . . . . . . . . . | 35·834 | | |
| Organic Matter, . . . . . . . . | 30·267 | 84·465 | |
| Fixed Salts, . . . . . . . . . | 5·567 | 15·535 | |
| Uric Acid, . . . . . . . . . . | 1·000 | 2·790 | |
| Urea, . . . . . . . . . . . . | 11·001 | 30·699 | |
| Extractive, Albumen, and Ammoniacal Salts, . . . . . . . | 18·266 | 50·976 | |
| Chloride of Sodium, . . . . . . . | A trace. | A trace. | A trace. |
| Sulphuric Acid, . . . . . . . . | 2·296 | 6·407 | 41·242 |
| Phosphoric Acid, . . . . . . . . | 1·142 | 3·168 | 20·513 |

seventh, and eighth days of the pneumonia were each carefully examined with the tartrate of copper and potash, the sulphate of copper and potash, and the fermentation tests, no unequivocal indications of the presence of sugar could be detected.

The *post-mortem* examination of this patient showed that the left lung was solidified throughout its entire extent, a condition quite sufficient to account for the fatal character of the disease; for one of the most important circumstances, I believe, which influences the result in pneumonia, is the amount of lung involved. This is one of the great points to be attended to in tabulating cases of this disease for statistical purposes; and it is one of those the neglect of which has rendered almost all the statistical observations on this malady of no worth, because the authors have contented themselves with simply noting cases as pneumonia, without specifying accurately the extent of lung inflamed. You may remember that in a former lecture I compared cases of pneumonia with those of severe burns. There is a difference among practical men, as most of you are, no doubt, aware, as to the mode of treating severe burns, some advocating a plan wholly antiphlogistic, and others recommend the very opposite of this, while a third party adopt the mean between these two extremes. Now it would obviously be not useless, but even absurd, to compare the results of these different methods of treatment, without at the same time taking into consideration the extent and severity of the burn in each particular instance; for every one knows, that a burn involving one-fourth of the entire surface of the body, is far more likely to prove fatal than one affecting only one-eighth of that surface. So in cases of pneumonia, the greater the extent of lung inflamed, the more difficult will it be to overcome the disease, whatever mode of treatment be adopted. Each plan will be the more successful, the smaller the amount of lung involved. Fortunately, in the generality of cases of pneumonia, not more than a third of one lung is in a state of inflammation. But in the case under consideration, not only was the whole of the left lung solid, but a considerable portion of it had advanced into the stage of purulent infiltration, a purulent fluid filling up the air-cells and finer bronchial tubes; and if this patient had lived a little longer, portions of this lung would, no doubt, have broken down.

The kidneys were in an early stage of chronic disease, the epithelium generally being opaque and crumbling, and many of the uriniferous tubes stripped of their epithelial lining, while in the cortical substance there were several cysts visible to the naked eye. The liver was in an early stage of cirrhosis; and there was some degree of morbid opacity of the mitral valve. This morbid condition of the liver and heart exercised an unfavorable influence in the case; but what tended principally to the fatal result was the disease of the kidneys, which there is every reason for supposing did not eliminate effete matters from the system, as they usually do in the state of health.

Case LXXXIII. (vol. xlii, p. 189.) Let me now speak of the case of pneumonia which recovered. It was that of William Davenny, æt. 20, a young man who was in the hospital about the same time as the last patient. This man, though young, was of very intemperate habits, the account of him being this: "He was born in London, and had lived in town nearly all his life, and is a shoemaker by trade; his habits are very dissipated, and he has made it a constant practice to get drunk whenever he has had an opportunity, ever since he was very young. He has never had any illness before the present. On the evening of January 17th, 1854, he went to a public-house to spend the evening, and drank beer till he was nearly drunk; he then played at skittles till very late, and got very hot; having finished, he walked home in the rain, his clothes became quite wet through, and he went to bed without changing his wet shirt. About three o'clock in the morning he was awoke by a severe pain in the left side of the chest and great difficulty of breathing. He began to cough, but there was no expectoration. He felt very cold, but did not shiver; and an hour or two subsequently he became very hot, and sweated a good deal. On the following day, January 18th, he kept his bed, and had alternate shiverings and sweats, and entirely lost his appetite; and from this time the pain in the side and the shortness of breath continued to get worse daily, until his admission into Sutherland ward on the 21st January, 1854." Here, then, we may fairly take January 18th as the date of the commencement of the pneumonia; and, therefore, the day of his admission would be the fourth of the disease. "He was able to walk to the hospital," continues the report; "his skin is very hot and dry, his tongue is covered with a white fur; he coughs much, but the sputa are not very tenacious; and when he attempts to take a deep inspiration a pain catches him in the left side of his chest." We then ascertained that there were all the signs of pneumonia of the lower lobe of the left lung, and that the inflammation had spread through the substance of this lobe, for there were all the indications of solidification in front as well as behind. Free counter-irritation was at once applied to the chest by means of turpentine stupes, and he was ordered six-drachm doses of the *liquor ammoniæ acetatis* every three hours, with milk diet and beef tea, but no wine was allowed.

The next day the pulse was 120, and the respirations were 48 a minute, and he was now ordered half an ounce of wine every third hour. On the sixth day of the disease he began to sweat, and it is noted in the report of the case, "pulse 104, respirations 32; he has been in a constant sweat all day." On the seventh day he still sweated copiously, his pulse was 76 and his breathing 24. On the sixth and seventh days there was well-marked large crepitation, taking the place of the bronchial breathing and bronchophony, which had previously existed, and from this time the patient steadily progressed towards recovery. On the eighth day of the disease the pulse and breathing were 76 and 28 respectively; on the ninth day the numbers were 68 and 28; on the tenth day they were 84 and 28; on the eleventh 68 and 24; and on the twelfth 72 and 20. With this gradual diminution in the frequency of the pulse and respirations there was a marked improvement in the physical signs,—the large crepitation gave way to pure breathing, and the dulness on percussion disappeared. At one time there was a slight amount of pleuritic effusion, but this speedily became absorbed under the influence of iodine paint. On the sixteenth day of the disease the report is as follows: "Feels quite well; there is still a little dulness on percussion over the base of the left lung behind, but the breathing is quite clear and vesicular." On the seventeenth day from the commencement of the attack this patient was discharged.

It is very possible in these cases that the lung remains in a swollen or œdematous condition for some time after the signs of pneumonia have disappeared, for it often happens that several days will elapse after the breathing has become perfectly pure before the dulness on percussion and increased vocal vibration will have entirely vanished.

In this case a marked improvement took place on the seventh day of the disease; indeed, there was something very like a *critical* sweat. Observations, too, were made on the urine, though, I am sorry to say, they were not quite so regular and accurate as in the other case. On the fifth day the urine contained no chloride of sodium, but on the tenth day that salt had returned in great abundance. Nothing can be more curious than this fact, which seems to be a constant occurrence in pneumonia; so much so, indeed, that the old doctors (and now the modern ones), who were in the habit of examining the *evening and morning stale*, could, had they been aware of this circumstance, and known, of course, that the case was one of inflammation of the lung, have predicated almost with certainty the condition of the patient—whether the disease was progressing favorably or otherwise.

Let us now briefly consider why one of these patients recovered, while the other died, or, in other words, the causes which tended to bring about the fatal termination in the one case, and the favorable in the other.

The circumstances which materially influenced the fatal result in the first case were, 1st, the extent of the disease; 2d, the age of the patient; and 3d, the state of the general constitution. In addition to these, the patient was one of those individuals who have undergone a great amount of wear and tear; and, moreover, his kidneys, heart, and liver were all damaged.

In the second case, although the lung was more extensively affected than ordinarily, the extent of pneumonia was less than in the first, and there was no disease of any other important organ. In both, the treatment was essentially the same; but the younger patient was, perhaps, better attended to than the older one, and was not quite in so depressed a condition.

With respect to the modes of termination of pneumonia generally, let me say a few words. When recovery takes place in this disease there is always, I believe, some kind of *critical* evacuation, either by sweats, or by urine, or by the free discharge of a purulent fluid from the bronchial tubes, or by pulmonary abscess. Some physicians look upon the herpetic eruption, which sometimes appears upon the lips in this disease, as of a *critical* nature; but I am disposed to think that it is rather a symptom of the disease, because it almost always occurs very early. Still, when you meet with it, you may regard it, I think, as a favorable sign; for most of the cases in which I have seen it, have done well.

One can say very little with respect to the intimate nature of these phenomena as spontaneous efforts of the constitution to throw off the disease; but we find this so often the case in other maladies—acute rheumatism and gout, for example—that I think in our treatment we should adopt that method which would give the best aid to this object. I am not prepared to hold the seventh, fourteenth, and twenty-first days as *critical*, as was the custom of the old physicians, but am contented with believing that there is a tendency in the disease to terminate on or near these days. Looking to the recorded cases of this disease, a *critical* evacuation by the urine is by far the most common; from the sixth to the tenth day this excretion being generally considerably increased, and having a tendency to deposit lateritious sediments. The *critical* evacuation next in order of frequency is that by sweating; and if treatment were adopted to favor the secretion of the skin, this would be far more common, I believe, than has hitherto been observed. A *critical* evacuation by the free discharge of purulent fluid from the bronchial tubes is not common, but I have seen it so profuse as to give rise to the supposition that an abscess had formed in the lung, and had emptied itself through a bronchial tube, when such was really not the case.

When death occurs in pneumonia, it is either by the exhaustion, consequent upon a very severe febrile disease; by breathlessness—apnœa—from the large amount of lung inflamed; by gangrene; or by abscess: but by far the most common mode of death is that by exhaustion, as occurred in the first of the cases we have just been considering.

There are certain circumstances which influence the prognosis in this disease. These you must carefully consider before you pronounce an opinion as to what will be the probable issue of any given case. The patient's life may be a highly important one, and his friends will expect you, as his medical attendant, to state decidedly (but you will do so cautiously) what you believe will be the most probable result of the illness. Now the circumstances which chiefly influence the prognosis are—first, the age of the patient; second, the extent of the disease; and from these two alone you may form a pretty accurate conclusion. But you should also pay due attention to the third point, which is the patient's general constitutional condition.

It has been fairly made out that there are two periods in life in which pneumonia is very fatal; these are from early infancy to the age of five years, and the advanced periods,—from seventy years and upwards. The great fatality of pneumonia in very young children and infants, is explained partly by the more feeble powers of vital resistance at that early period of life, and partly by the fact that at this age it is seldom uncomplicated, and is almost always combined with bronchitis; and there can be no doubt that bronchitis in itself is at all ages a much more fatal disease than pneumonia.

According to French statistics, a death from pneumonia was rare between the ages of five and fifteen. From fifteen to thirty, in 116 cases, there were eight deaths; from thirty to forty, the number of fatal cases amounted to one-seventh of the whole; from forty to fifty they were one-sixth; from fifty to sixty, one-fifth; from sixty to seventy, one-sixth; and above the age of seventy, as many as eight-tenths of those attacked died. If, then, your patient be between fifteen and thirty years of age, and if the amount of lung involved be not considerable, the chances of his recovery are good; but if the whole of one lung be inflamed, or the half of both lungs, then you must look very gloomily at the case; while if the disease affect only the half of one lung, or only a third, or still less, you must take the more cheerful view, and, in accordance with that view, give a more favorable prognosis. Much will depend on the constitutional powers of the patient, his power of vital resistance; and the existence of any previous damage to any vital organ will, of course, militate considerably against a favorable termination. Something, too, may be due to what is termed *epidemic constitution;* for it cannot be doubted that typhus fever, erysipelas, and some other diseases, are much more fatal in some seasons than in others, and this may, to a certain extent, obtain in the case of pneumonia.

Another circumstance which you must take into consideration as influencing the prognosis, is the plan of treatment adopted. I believe that in this disease you can do much more by treatment to hasten a fatal result, than to bring about a favorable termination, and that the interference of the physician should be with the view of assisting nature in getting rid of those materials, the retention of which in the system appears to produce the disease. This may be done by promoting the secretions of the skin and kidneys; at the same time paying due attention to the condition of the digestive organs and bowels. Your aim should therefore be to induce free sweating, to apply counter-irritation to the chest, and to exhibit mild aperients. Finally, the patients should always be supported by the liberal, but well-regulated, administration of good nutritious food.

If blood be taken, it should only be with the view of relieving pain, and for this purpose the application of a few leeches over the painful spot is the most successful plan; but when the dyspnœa is extreme, it is sometimes expedient to take a little blood from the arm. Those of you who are in the habit of attending my hospital practice will bear witness how very rarely I have recourse to this proceeding. To bleed, or not to bleed, is often a question in pneumonia, and one which I earnestly recommend you, as a general rule, to decide in the negative rather than in the affirmative.

Let me conclude what I have to say on this subject, by calling your attention to a report which I have before me, and which Mr. C. Macnamara has been kind enough to draw up for me, of all the cases of pneumonia which have occurred under my care in the hospital from 1840 to 1853, and now corrected up to 1859. The cases are in all 78, and they have been taken just as they have been noted down in the Case-books. They may be arranged into two periods: the one from 1840 to 1847, the other from 1847 to 1859. In the former of these, which may be termed *the period of reducing treatment*, the remedies employed were, bleeding, the application of blisters, the exhibition of tartar-emetic, and the more or less free use of mercury; while in the latter, which may be styled *the period of supporting treatment*, the patients were treated very much in the manner which I have detailed to you in this and other lectures on this subject, though in a few cases calomel and opium were given. Now, the total number of cases, as I before mentioned, was 78, and of these 10 were fatal.

Remember that I am now merely stating the leading points in the report, for time will not permit me to enter into any lengthened detail. Of the seventy-eight cases, twenty-five occurred in the first period, and four of these proved fatal, or about one in every six. In the second, the number of cases was fifty-three, and in these there were only six deaths, or about one in nine; fairly leading, I think, so far as we may judge from the relative mortality in these two periods, to the inference that the supporting plan of treatment is more favorable (certainly not less favorable) in its results than the severer measures which are frequently had recourse to in this disease.

## LECTURE XIV.

### ON THE THERAPEUTICAL ACTION OF ALCOHOL.

GENTLEMEN,—I have selected for to-day's lecture the case of a little girl poisoned by alcohol. I fear few of you have seen this patient, chiefly those who have been about the hospital during the summer months, as she was admitted into the wards, and indeed died, a few days before the commencement of the winter session. The subject of poisoning by alcohol is a most important one, and cases of it are rare; on this account alone it deserves special consideration, but it also enables me to make some observations explanatory of the grounds upon which a good deal of the practice in acute disease, which you will see carried out by me in the wards of this hospital, is founded.

Some of you coming into the wards for the first time, will doubtless be surprised at seeing that a good deal of wine and brandy is administered to many of the patients. And you must feel inclined to exclaim, "This seems very heterodox practice, and altogether different from that which I had been led to expect, or am accustomed to witness." When you further examine the patients who are under this treatment, and find that many of them have a hot skin and a rapid pulse, you may be even disposed to think that such practice looks like an adoption of the principle, *similia similibus curantur;* but when you look more closely into the matter, you will find that no such absurdity holds its place here. I have been on the look-out for a case upon which I could base a few remarks upon the administration of alcoholic fluids, especially in disease; and happening to be in the hospital when the patient to whom I have alluded was brought in, the house-physician came to tell me that he had just admitted a case of marked interest, tauntingly replying to my inquiries as to its nature, that it was one of poisoning by a favorite remedy of mine, alcohol. These circumstances, together with many instructive points exhibited by the patient, have induced me to embrace this opportunity of directing your attention to the subject of the administration of alcohol, both as a poison and a therapeutical agent.

Alcohol, in some form or other, is a remedy whose value can scarcely, I think, be over-estimated, and one upon which, when carefully administered, I rely with the utmost confidence in a great number of cases of disease which are at all amenable to treatment. You must bear in mind that there are very many instances of morbid conditions, which will come under your notice hereafter when you come to practise for yourselves, which are not in the least degree under the influence of any remedies with which we are at present acquainted, and in which the subjects of them will die, do what you will; while, on the other hand, there are many cases in which recovery will take place, no matter what treatment you adopt, even if you give the patient nothing but cold water. Hence, in studying the effects of a remedy in any disease, you must always carefully separate from the data upon which you are to found your conclusions, these two classes of cases, viz., those which will recover without any treatment at all, and those which will most assuredly die in spite of every effort to save them.

CASE LXXXIV. (Vol. xlvi, p. 114.) The case, then, to which I wish to direct your attention to-day, is that of a child named Mary Keller, æt. 3, who was admitted into the hospital about half-past three o'clock on the afternoon of September 19th, 1855. We were informed that a quantity of *gin*, stated not to have exceeded "half-a-quartern," (2½ oz.) had been administered by her mother, about nine o'clock on the morning of the same day. The motive of the mother in thus dosing her offspring with gin—whether it was merely a drunken frolic, or whether it was induced by some bad intent—could not be ascertained. Very soon after the dose, the child fell off into a deep sleep; she was then put into a close bed, and some time afterwards the parents, finding that they had great difficulty in arousing her, became alarmed, and at length brought her to the hospital, about *six hours* after the administration of the gin.

Now the circumstances which here strike one most are the great rapidity with which the symptoms manifested themselves, and their steady increase from the time of the administration of the poison, until the patient came under observation.

Let me read to you the description which the house-physician, Mr. Workman, gave of the little patient: "The child was well-grown and of healthy appearance; she lay insensible in her mother's arms, as if asleep; the pulse was good, the body and extremities warm, and the eyes presented a natural appearance." As the history of the case showed that these symptoms followed almost immediately upon the child's swallowing a certain quantity of gin, the first indication for treatment plainly was to endeavor to extract from the stomach any alcoholic fluid which might yet remain there. An emetic was therefore at once administered, but was swallowed with considerable difficulty; and as vomiting did not follow soon, tickling the fauces was tried. But even then the little patient merely coughed and struggled, and did not effectually vomit. "Cold water was now poured in a stream upon her head, and as the insensibility of the patient was extreme, mustard poultices were applied to the body, and the feet were well slapped, but all without effect: after this, a mustard bath was tried. The most effectual excitant," continues the report, "was the natural one of slapping the posteriors, to which the child manifested her objections by crying loudly, and by kicking, *but with the right leg only.*"

Here was a new symptom, demanding our special attention in the investigation of the nature of the case. The patient could move the right side only, the left arm and leg being paralysed, and the arm more so than the leg. Some warm water was now injected into the stomach, but when drawn off it did not emit any odor of gin, neither did the breath at any time smell of this fluid; but inasmuch as six hours at least had elapsed between the administration of the alcohol and the time of the child's coming under observation, there was good reason for believing that the spirit must have been entirely absorbed. At half-past six o'clock the same evening, another new symptom manifested itself, which consisted in an epileptic fit. "The child first opened her eyes, which had been previously closed, as if about to recover her consciousness, and coughed and vomited a little; the muscles of the left side of the body and face then began to twitch convulsively, and the right side also shared in the convulsions, but only in a slight degree." This looked very much as if the peculiar influence which produced the convulsions, whatever its precise nature, was seated in the same side of the brain, and was most probably the same as that which gave rise to the paralysis. "The convulsions lasted about three minutes, and the child then fell again into her previous quiescent state; but this was every now and then interrupted by a similar convulsive paroxysm, which recurred throughout the night to the number of twenty to thirty. The bladder became distended, and in the course of the evening was emptied by means of a catheter. The urine which was drawn off was clear and transparent, and healthy. Five grains of calomel were now given, and this was followed up by a dose of castor oil."

On the 20th the report is as follows: "The child lies quiet, taking no notice of anything except when roughly pulled about; she has passed plenty of urine, but the bowels have not been opened; the left arm is still paralysed.

"2 P.M.—The condition of the patient is much the same: there has been no return of the fits since nine o'clock this morning: the left leg is but very little paralysed, and the arm also is less so than when last reported: pulse good. The child has been fed with milk and arrow-root.

"10 P. M.—About half an hour after the last report, another fit, but a very slight one, occurred; with this exception, there has been no return of the fits. She has vomited everything she has taken during the day: the pulse is weaker and more rapid, and the patient is evidently becoming exhausted. Ordered to take a teaspoonful of wine every second hour, with a little beef tea, and to have a castor oil enema administered at once."

On the 21st, the following report was made: "The aspect of the patient has altered much, and for the worse; she still lies as if asleep, with her eyes half-closed but deeply sunken in their sockets, and the whole face seems swollen; the sickness is much less than it was yesterday; the breathing is quiet; pulse 156. The pupils are equal, but oscillate a little. The patient exhibits some degree of consciousness, and opens her mouth when food is presented, and once she appeared even to recognize her parents; still she usually lies in a torpid state, occasionally turning in bed with a fretful moan, and tossing her right arm across her face; the left arm she never moves."

On the 22d this note was made: "The symptoms continue much the same as yesterday: the vomiting has ceased, and there has been no return of the fits, but the bowels have not been opened. Pulse 140; respirations, 30.

"10 P.M.—The patient is gradually sinking; the pulse and breathing are exceedingly rapid and irregular, but the body continues warm. She swallows readily." The patient continued sinking throughout the night, and died at half-past nine on the morning of the 23d, about four days after the administration of the gin.

The body of this child was most carefully examined after death, but we were unable to detect any abnormal anatomical condition of either the white or gray substance of the brain, excepting an extreme degree of pallor. This state of anæmia is the very opposite to that which the notions commonly entertained on this subject would lead us to expect; for, as most of you are doubtless aware, coma and convulsions are generally regarded by authors—most incorrectly in my opinion—as dependent upon a congested state of the brain—an over-distended state of the cerebral capillaries. In this case the brain generally was so pale that all those who were present at the examination were much struck by it; but there was no alteration in its consistence, though being that of a child, it was of course softer and more watery than the brain of an adult. There was no effusion into the cavities of the brain, nor any condition by which the coma could be accounted for by way of pressure.*

The thoracic viscera appeared quite healthy; and I ought to have told you that we had several times carefully examined this child during life, with the view of ascertaining the condition of the pulmonary vascular system (for there are many who imagine that alcohol retards the circulation, and thus gives rise to congestion). On these occasions we always found the breathing pure and vesicular throughout the lungs, except in the lowest part of the inferior lobes posteriorly, which we concluded were much congested, with more or less of an œdematous condition. This state was no doubt due to the dependent position of these portions of the lungs. You are always likely to meet with it in cases in which the vital and nervous powers are materially depressed, as for instance, in fever, in which disease the patient almost always lies upon his back. The effect of posture may be well illustrated after death, for if the body have been lying some time on the back, you will find the posterior parts of the lungs more or less congested; but if you now turn it, and allow it to remain for some time with the belly downwards, the blood will gravitate to the most dependent parts, and the congestion will be transferred to the anterior parts.

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* I very much regret that the specific gravity of different portions of the brain was not taken.

This tendency to congestion, exhibited in these cases during life, evinces a feebleness of the nervous influence in the capillary vessels, so that the blood obeys the laws of gravitation more readily than it is permitted to do in health. Thus, in weakly women and in dropsical patients, you will frequently find that the lower limbs *pit* upon pressure, and exhibit the usual characters of œdema; and this condition, you will generally learn, is aggravated towards the evening. These phenomena result from the insufficiency of the forces by which the circulation is carried on in the capillary system, and which, in such low states of vital power, are incompetent to prevent the blood from obeying the ordinary laws of gravitation.

But to proceed with the results of the post-mortem examination. Nothing could be found amiss with the stomach or intestines; the liver looked healthy, and the kidneys tolerably so, but about these last there was some difference of opinion. For my own part, I thought they closely resembled the kidneys of persons who die of that form of dropsy which so frequently follows scarlet fever, the medullary portions being exceedingly red, while the cortical substance was unusually white; this last-named condition resulting apparently from an undue accumulation of the renal epithelium in the uriniferous tubules, and chiefly in their convoluted portions. But you ought never to place reliance upon the condition of the kidneys as determined by naked-eye observation; and whenever there is the least possible doubt, you should always carefully investigate the real state of these organs with the aid of the microscope.

In the present instance, the results of a microscopical examination showed that the renal epithelial cells generally, although not existing in any undue quantity, and still adhering intimately to the basement membrane of the tubular walls, contained in their interior much fatty matter, and that the kidneys were good specimens of that which has been designated by Dr. George Johnson "*the mottled fatty kidney.*"

To what this condition of the kidneys was due, it is exceedingly difficult to say. Two questions suggest themselves. Was it a condition of prior existence, and did it contribute to increase the symptoms excited by the alcohol, or to oppose the recovery of the patient? or was it an effect of the administration of the alcohol? Those of you who are acquainted with the peculiar affinities of fat and alcohol, will find here a fair field for speculation; for my own part, I must be content with simply calling your attention to the clinical facts. Were I otherwise disposed, time would not permit me to enter into so wide a discussion. Let me remark, however, that it seems highly improbable that such a state of kidneys as I have described could be produced in so short a time as that which had elapsed since the gin was taken. On the other hand, I think it not at all unlikely, as the child was pale, and had no doubt been always badly fed, although she was not decidedly unhealthy-looking, that slow disease of this kind may have been gradually developing itself. You will perhaps remember that a state of kidney somewhat analogous to this is frequently found in London cats, and it does not appear improbable that those London children, of the lower orders, who are reared in the same sort of darkness as the cats, while they are also irregularly fed upon unwholesome food, and are continually breathing an impure atmosphere, may exhibit the same proneness to this peculiar morbid condition of the renal organs as their feline companions are so apt to do. Assuming that this is the correct explanation of the condition of the kidneys, we at once obtain a clue to unravel some of the phenomena which this patient exhibited, more especially the continuance of the prostration and coma after the other signs of poisoning by alcohol had entirely disappeared.

Let me now direct your attention to some of the practical information which we may extract from this highly interesting case; and it will be convenient if I at once arrange, under three heads, the points most worthy of your notice. These are:—

1. The manner in which alcohol finds its way into the system, and the organs or textures on which its influence is most directly exerted, and for which it has the greatest affinity.

2. The mechanism, so to speak, of its action on the body, and how it may operate as a poison.

3. How far it may be used as a therapeutic agent, and the manner in which it may be employed as such, whether in acute or chronic diseases.

I. Perhaps the most remarkable phenomenon exhibited in this case consisted in the extreme rapidity with which the symptoms were developed after the ingestion of the gin. How is this to be explained?

If you will take the trouble to look to the histories of various cases of poisoning by alcohol, reported in books on toxicology (and I know of none better than that of Dr. Christison) you will find that there is nothing peculiar in this speedy occurrence of the symptoms, where persons—even adults—have swallowed a considerable quantity of spirit; indeed, that it may be considered as the rule, that after the ingestion of alcohol its peculiar effects show themselves with remarkable rapidity.

Let me briefly refer to two or three of the cases illustrative of this subject, which you will find recorded in Dr. Christison's work. One is that of a man who stole a bottle of whiskey, and finding that he was in danger of being detected, but being unwilling to part with the stolen spirit, at once drank off the entire contents of the bottle; but he would scarcely have done so had he been aware of the remarkably rapid manner in which alcohol produces its effects; almost immediately he fell into a state of coma, which continued until death took place, about *four hours* after he had swallowed the whiskey. Another case is that of a boy who drank a large quantity of raw whiskey, and immediately after taking it, fell into a state of coma, upon which, in the course of two hours, tetanic convulsions supervened. The stomach-pump was now employed, and the greater part of the whiskey withdrawn, when the lad recovered his consciousness for about a quarter of an hour, but he afterwards relapsed into a state of stupor for the whole of the day. A third case is that of a lad fifteen years of age, who laid a wager that he would drink sixteen ounces of whiskey, and then, to show how little effect it would take upon him, would walk up and down the room a certain number of times. This feat he performed, and for some time after appeared none the worse for it; but, about half an hour subsequently, he walked out into the open air, and whilst in the act of putting his hand into his pocket, he became perfectly insensible, and was, of course, unable to take his hand out again. From this moment he gradually became more and more comatose, and at length died, about *sixteen hours* after having drunk the whiskey.

These cases recorded by Dr. Christison correspond very much with that which I have brought before you to-day, and they show how rapidly the symptoms peculiar to poisoning by alcohol supervene upon swallowing a large dose of that fluid. The principal reason of this seems to be, that alcoholic liquids, especially spirits, such as whiskey, brandy, &c., are within certain limits, extremely easy of digestion. In other words, the mode in which the digestion of alcohol takes place is by no means complicated, but consists rather in the simple process of absorption. A man drinks a glass of brandy and water; what becomes of it? It passes down into the stomach, and is at once absorbed directly by the bloodvessels, which, as you know, ramify so freely in the walls of this viscus. If, however, the quantity of spirit and water taken be very great—say a couple of pints or more—and if a short time be occupied in drinking it, then a part passes down into the intestines, but the greater part still remains in the stomach, and finds its way, by a process of *endosmosis*, directly into the bloodvessels of the mucous membrane, with which it is in contact.

The digestion of lean meat is the next in point of simplicity to that of alcohol. After undergoing proper mastication in the mouth, and a certain amount of admixture with the salivary secretion, meat enters the stomach, where it is subjected to the solvent action of the gastric fluid; and, having been thus dissolved, whilst at the same time it undergoes a certain amount of chemical change, it for the most part passes immediately into the bloodvessels of the stomach. But the process, *preparatory* to digestion, which occurs in the stomach in the case of meat, has nothing analogous to it in that of alcohol; the latter being when swallowed, already in a form which admits of its complete and ready absorption by the gastric bloodvessels. The fact that there is no need for any previous process prior to absorption, explains the rapidity with which the alcohol enters the circulation, and its immediate influence in producing the symptoms, as observed in our patient and in those whose cases are related by Christison.

It was long believed that alcohol exercises some special influence on the sentient extremities of the nerves of the stomach and intestines; and that it was not absorbed into the blood at all. But of its actual absorption, when swallowed, there is abundant evidence, as well as of the almost incredible rapidity with which it is diffused through the circulation. An important proof of this absorption is furnished by the fact that very soon after swallowing the spirit, especially when taken on an empty stomach, fumes of alcohol are perceptible in the breath expired. And it is a highly interesting fact, which we have observed very often in the hospital, that in cases of great exhaustion of the system, when it stands much in need of alcohol, this does not occur; the spirit undergoing certain chemical changes in the body, and not passing out through the respiratory organs in the form of alcohol.

II. The effects produced by the free ingestion of alcohol are all referable to the *nervous system.* The prominent symptom is *coma.* If you institute a careful search into all the cases of poisoning by alcohol upon which you can lay your hands, you will find that in every one of them a greater or less tendency to coma was present. To coma are sometimes added *convulsions,* generally of a clonic, but occasionally of a tetanic character: in some cases the coma is preceded by *delirium,* in others it is followed by *paralysis.* When the fluid swallowed contains a very large proportion of alcohol, *vomiting* is apt to occur; the alcohol in these instances probably acting as a direct irritant to the mucous membrane of the stomach, which becomes red, vascular, and congested, as if it had been subjected to the action of an irritant poison. Generally speaking, however, symptoms referable to the *digestive* organs do not take a prominent place among those induced by the ingestion of alcohol.

And here I should remind you that ale, porter, wine, brandy, gin, &c., contain alcohol in very different proportions. With these proportions you ought to be familiar, for this is a point upon which, as a matter of dietetics, you will be hereafter almost daily consulted.

But to return to the symptoms due to the ingestion of alcohol. The principal effects, as the symptoms in the cases which I have detailed show, depend on the influence exercised by alcohol on the nervous system. Alcohol is, as you are aware, a hydrocarbon; and almost all hydrocarbons have a marked affinity for the nervous system, as compared with the other structures of the body; and it is upon the nervous centres that alcohol exerts its primary influence. At first its action is simply to augment the generation of the nervous power. When this increased action of the nervous system is kept within certain limits, the effect of the alcohol is beneficial; but when carried beyond a certain point, its action is injurious and deteriorating, the spirit at length impairing, and ultimately destroying the nutrition of the nervous matter.

Evidence of the tendency of alcohol, when taken in excess, to damage the nutrition of the nervous matter, is to be found in the peculiar tremblings and impaired mental power, which are commonly present in cases of delirium tremens. These tremors are solely indicative of too great waste of nerve-matter, and they often occur under circumstances in which there has been an undue expenditure of nervous power, such as great mental anxiety or fatigue, without any recourse having been had to the use of alcoholic liquids.

In persons who have died after a long course of intemperate indulgence in fermented liquors, we find a further proof of the damage done to the nervous system in the shrunken brain, the wasted convolutions, the increased amount of subarachnoid fluid. The use of alcohol in too great quantity—in other words its abuse—causes a wasting and degeneracy of the brain, both of gray matter and fibrous matter. There is no congestion unless the death of the patient has been immediately preceded by a prolonged struggle, or by convulsions; and the subarachnoid fluid merely follows the brain-shrinking, and serves to fill up the space which that shrinking had left vacant.

Of the various unfavorable effects which alcohol is capable of producing, it behooves the practitioner to observe that inflammation of internal organs is not one. It is true that a strong alcohol, applied directly to the mucous membrane of the stomach, or the conjunctiva of the eye, may cause inflammation of those membranes. But alcohol absorbed into the blood will not excite inflammation of the lungs, or the heart, or of the liver,[1] or kidneys, or the bowels, nor even of the brain. I have never seen, nor heard, nor read of any authentic case in which inflammation of any of these organs was attributable to the ingestion of alcohol. Look at the case of our little patient; there was not a particle of inflammation in her body, not even in the mucous membrane of the stomach. The brain was anæmic, but altered in its consistence, showing that the poison acted on the nervous matter by attraction from the blood, and illustrating how coma, convulsions, and paralysis may all occur independently of hyperæmia, or of inflammation.

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[1] Cirrhosis of the liver is an atrophy, not an inflammatory disease of that organ.

I apprehend, that in all cases of brain affection from alcohol, the symptoms are due, not to congestion nor to inflammation, but to a simple poisoning of the nerve-fibre and nerve-cell, by the spirits. It is the same with chloroform and sulphuric ether, when inhaled. They act directly on the nerve-fibre and nerve-cell, and not by producing congestion. If you soak a living nerve in chloroform, for a certain time, it will refuse to propagate the nervous force under chemical, mechanical, or electrical stimuli.

I think, then, we may discard the popular prejudice, that alcohol causes inflammation, and that therefore it ought not to be given in inflammation.

There is another notion which certain objectors to the use of alcohol are fond of adducing, namely, that if alcohol causes exaltation of nerve-force, that is followed by a corresponding amount of depression. This, I am sure, is a fallacy due to the comparison of a patient's feelings when under the immediate influences of the spirit, or other alcoholic fluid, with the sensations which accompany the absence and the want of its exciting influence. There is no evidence to show that any physical depression is induced as a sequel to the alcoholic excitement, that any increased waste goes on, or that the nervous power is at a lower ebb than *before* the administration of the alcohol, if it have been given in proper quantity. When alcohol has been taken in *inordinate quantities*, then the feeling of depression is the greater by reason of disturbed digestion; but even under these circumstances evidence is wanting to demonstrate the existence of a distinct physiological depression of vital power, unless by its repeated over-use the nutrition of the nervous system be impaired.

In its further effects upon the body, alcohol taken carefully, increases the animal temperature; it also strengthens the action of the heart; and, when administered under proper circumstances, it reduces the frequency of the pulse. So far as it influences the nervous system, the action of alcohol is that of what is commonly called a *stimulant*, an unfortunate term, indicating a distinction without a difference. Other forms of food are likewise stimulant, but as they do not act directly and quickly on the nervous system, their exciting properties are not so apparent. In like manner, alcohol possesses its stimulating property because it is a form of aliment, appropriate to the direct nourishment of the nervous system, and to its preservation; and its special adaptation to this system gives it an immediate exciting power superior to any other kind of food. Alcohol is also fitted to uphold the calorifacient process, and therefore to protect the nervous and other tissues, which, without it, would be largely called upon to furnish fuel for the oxygen which supports that process. And for this purpose, alcohol possesses considerable advantages over most other hydrocarbonaceous substances, in consequence of the ready manner in which, when swallowed, it passes into the blood. Oil is a hydrocarbon equally with alcohol; but the digestion of the latter is a very different affair from that of the former. Oil, when swallowed, remains for some time in the stomach, where, however, it undergoes no change; it subsequently passes down into the small intestine, where it is subjected to the action of the pancreatic and other fluids, and having become converted into chyle, is absorbed by the intestinal villi. Thus the digestion of oil is a far more complicated process than that of alcohol. Hence, then, as a calorifacient form of food, as a promoter of the nutrition of the nervous system, and as admitting of easy and quick absorption into the blood, alcohol possesses a combination of qualities which render it of the utmost value in the treatment of disease.

Observe, I beg of you, that I use the term alcohol merely generically, to comprehend the various fluids which contain alcohol, in greater or less quantity. Alcohol is always, I presume, to be presented to the human stomach in a more or less diluted form. Pure alcohol, likewise called *absolute*, or *anhydrous*, can only be taken with extreme caution, and in very small doses. Its influence on the mucous membrane would be that of an irritant; that is, it would so disturb the physical condition of the elements of that membrane as to create hyperæmia, to interfere with its secretions, and consequently greatly to impair primary digestion. In the same way a large quantity of any alcoholic fluid quickly swallowed, will produce a similar effect, and will cause considerable disturbance. And we learn from this that the true method of administering these fluids is, to use a common phrase, by giving "a little and often." Fix upon a dose which you may think your patient's stomach will bear—from two to twelve, or even sixteen drachms—and give that more or less diluted with water or some other bland liquid, at such intervals as his state of vital power may seem to demand.

And if we look at alcohol in this point of view, and especially as a food peculiarly adapted for the calorifacient process, and admirably suited to shield the tissues against the influence of oxygen, under circumstances when substantial food adapted to the nourishment of those tissues cannot be taken, looking, I say, at alcohol in this point of view, it is plain that the quantity to be given is that which should especially engage the attention of the practitioner. You must give enough to keep up animal heat and to protect the tissues without embarrassing the stomach, and without allowing the fumes of alcohol to be perceived in the breath. All that is given over and above that quantity is wasted, so far as the support of the vital powers is concerned. Now we cannot as yet exactly measure the wants of the system, and it is only by trial and experience that we are able to adjust approximatively the quantity to be given to the wants of the system. For this reason I have adopted the plan of giving a precise dose at brief intervals, from two to twelve or sixteen drachms, diluted, just as you give so much calomel, or so much opium, at specified times.

III. Having now examined the poisonous effects of alcohol, and also explained to you its mode of action, let me call your attention very briefly to the particular forms of disease in which you are most likely to find alcohol useful for promoting the healing process by upholding vital power.

But here I think it necessary to guard myself against the imputation of encouraging the excessive and improper use of alcohol. No one who thoroughly appreciates the great value of this agent in the treatment of disease, will regard with indifference the abuse of it in health. And I will yield to no one in the desire to see this beneficent gift of God to man (so easily converted into a destructive poison, capable of weakening nervous power and of annihilating all the best qualities of his mind), employed with the care and moderation through which alone it contributes to the health of mind and body. It is curious, indeed, to observe, that with the exception of the very lowest races of men, there is scarcely one of the various tribes with which this earth is peopled, which has not discovered for itself some method of preparing an alcoholic liquid; showing, I think, the existence of an instinct in human nature for such a food as alcohol.

Alcohol may be employed in all those diseases in which a *tendency* to depression of the vital powers exists; and there are no acute diseases in which this lowering tendency is not present. Many such maladies will, doubtless, get well without the interference of art; cases of pneumonia, and of fever, for example, will frequently recover without the employment of any medicines. But take any case of pneumonia thus left to follow its own course, and after eight or ten days you will always find your patient more or less emaciated, and in a much more prostrate condition than on the accession of the malady. If, however, in such a case tartar emetic had been exhibited, or bloodletting practised, how much more depressed will be his condition! Hence the great importance of upholding your patient's powers while the natural processes of the disease are taking place. In pneumonia a peculiar effusion occurs into the air-cells, which after a while become filled, the lung itself becoming dense and solid. For the recovery of the patient all this effusion has to be absorbed, and the air-cells restored to their original condition. The processes which nature has to perform in the cure of this disease are highly complicated, and no one, I apprehend, will assert that we posess any drug which by its direct agency on the system can effect this object. In the accomplishment of these changes there is a considerable expenditure, as it were, of nervous force and of blood; and therefore, we should supply to the system a kind of food which, while it is easily assimilable, is at the same time capable of upholding the nervous power, and of maintaining the animal heat. And such a food is alcohol, which, as I have already pointed out, is assimilated in the easiest manner by a simple process of endosmosis, exercises a peculiar influence on the nutrition of the nervous system, and by its combination with oxygen in the body, supplies fuel for the maintenance of the animal temperature. When given in too large a quantity, alcohol passes out of the body unchanged in the air expired from the lungs; but when its amount is limited and proportionate to the real wants of the system, it is evolved in the form of carbonic acid and water, and promotes the secretions of the lung, the skin, and the kidneys.

The successful use of alcohol in the treatment of disease depends very much on its *mode of administration.* Alcohol should be given in such doses as experience shows are assimilated with the greatest ease. Let me illustrate this by an example. If two individuals drink a bottle of brandy each, the one in the course of one or two hours, the other in doses of half an ounce every hour until the whole is consumed, the effect of the alcohol in the two instances will be very different. The former person will have become more or less intoxicated; or, in common language, "*drunk*," unless he have been long addicted to large potations of brandy; but the latter will suffer but little general disturbance, except that the heart's action will be somewhat excited, and certain of the other effects of the ingestion of alcohol produced, but there will be no delirium, drunkenness, or coma. This difference in the effect of the alcohol in each case arises from the *mode of administration.* A difference of a similar character is also observable in the influence of some other medicines; for example, opium.

In prescribing alcohol in disease, you should not order so much brandy or other spirit, to be given *daily;* but you should direct a certain quantity—say a teaspoonful or a tablespoonful—diluted with water, to be administered every *half hour*, every *hour*, or every *two or three hours*, according to the nature of the disease and the actual condition of the patient.

There are at the present moment several cases in the hospital, in which alcohol is being administered in the manner I have just pointed out, to some of which I may briefly refer.

Case LXXXV. James Booth, æt. 21, a baker, now in No. 4 ward; brought into the hospital laboring under typhus fever, which came on about a fortnight before his admission. He was immediately ordered to take *half an ounce of brandy every two hours*, while at the same time another form of stimulant, namely, ammonia with chloric ether, was also administered.

The case was not a bad one; but its progress shows the steady decline of the pulse under this plan of treatment. On admission the pulse was 90, the respirations being 30; the following day, the pulse was 86, the respirations being also 30; on the third day, the pulse was still 86; on the fourth day, it was 70; on the fifth, 72; on the sixth, 65; on the seventh, 65; and so it has continued at about the standard of health.

CASE LXXXVI. A lad, æt. 17, suffering from typhoid fever, the pulse on admission being 130, and the respirations 24. *Half an ounce of brandy* was ordered to be given *every three hours*, and likewise ammonia and chloric ether, as in the preceding case. The following day the pulse was 120, and the respirations 24. On the third day the pulse had risen to 125, at which rate it also remained on the fourth day. Many practitioners would have now said that the brandy was disagreeing with the patient, and was increasing the frequency of the pulse; but experience had taught me otherwise, and I came to the conclusion that the amount of alcohol supplied was insufficient; the dose was, therefore, increased to *half an ounce every hour.* And my expectations were realized; for on the fifth day the pulse had fallen to 120; on the sixth day, it was 110; on the seventh day, also 110; on the eighth day, it was 105; and on the tenth day, 96; and it subsequently continued gradually and steadily declining under the use of twelve ounces of brandy *per diem.*

In the treatment of pneumonia, I do not consider the administration of alcohol essential, but rather regard it in the light of an important accessory.

CASE LXXXVII. A case of this disease is now under my care in No. 4 ward, in which nearly the whole of the right lung was hepatized. In this case counter-irritation has been freely applied to the chest, and the patient on admission was put upon large doses of the *liquor ammoniæ acetatis,* while wine was also administered in doses of half an ounce every second hour. Under this plan the case has progressed favorably, and the pulse, which, on admission, was 90, has gradually decreased in frequency, so that on the ninth day after admission it had fallen to 76.

In bronchitis alcohol is not less useful and more necessary than in pneumonia. I give you from my case-book the following case in illustration of its administration in the former disease.

Case LXXXVIII. (Vol. xlix, p. 234.) Phœbe Skinner, a thin, spare woman, 45 years of age, married, with a large family, and poor general health, was admitted into King's College Hospital, March 13th, 1855, suffering from an attack of bronchitis.

The patient stated that she had been long subject to severe catarrhal attacks in winter time, and had been twice before in the hospital with bronchitis; that for the last three months she had suffered much from indigestion, so that she had been unable to eat, even sparingly, without suffering much pain in the stomach and chest afterwards.

Her illness had commenced a week previously, with great pain in the left side, accompanied by a frequent and hard cough, and frothy expectoration; at the same time her breathing became quick and difficult.

When admitted, her face was flushed, but of a dusky hue; she coughed frequently, expectorating a greenish frothy mucus, and complained of thirst, alternate flushings and chills, and aching in her limbs. Pulse, 120, feeble, and somewhat irregular; respirations, 36. Tongue coated. Crepitation was heard throughout the right lung, but most developed behind, and rhonchus and sibilus in the left.

Five grains of carbonate of ammonia were ordered to be given in decoction of senega every four hours, a blister to be applied to the left side, and turpentine stupes to the front and back of the chest.

The following day, March 14th (eighth of the disease), our patient continued extremely ill; towards evening the pulse became very rapid and feeble, and the respiration extremely embarrassed; expectoration was very imperfectly performed, and with the greatest difficulty, and there seemed to be some danger of suffocation. Half an ounce of wine had been ordered her on admission, every third hour; this was now changed for half an ounce of brandy every second hour.

By the morning of the 15th (ninth day), she appeared much relieved; the respirations were reduced to 27, the pulse to 108. The cough continued, and there was now a copious frothy expectoration, faintly tinged with blood.

On the 18th (twelfth day), the cough continued very troublesome, and expectoration, although freer, was still difficult. Wheezing and large crepitation were heard throughout the right lung, and the posterior part of the left. The patient seemed very feeble. Pulse, 102; respirations, 27.

On the 20th, although the pulse had fallen to 88, the respirations had increased in frequency to 36. In other respects she was much the same. Two grains of quina were now ordered three times a day.

From this time there was very little further improvement for nearly three weeks: the patient continued very weak, and the cough troublesome, with much frothy expectoration. She slept badly at night, and suffered from headaches, want of appetite, a furred tongue, pain in the side or epigastrium after eating, and attacks of diarrhœa.

The brandy was discontinued, and a little wine given. On the 1st of April the quina was changed for iron.

By the 10th of April the cough began to subside, and the expectoration greatly diminished.

On the 11th the patient was attacked with violent pain in the stomach, and headache, and had three epileptic seizures in quick succession. The same thing had occurred to her once before, when recovering from bronchitis.

From this time she improved steadily, and was discharged well on the 26th.

You have here an example of marked improvement in the state of the pulse, following immediately upon a greatly increased allowance of alcohol. Under it the pulse fell from upwards of 120 on the eighth day, to 108 on the ninth.

There was some peculiar state of nervous system in this patient, which seemed to explain her prolonged convalescence, and which was relieved by a discharge of nervous force, in the shape of three epileptic seizures. It is not likely, although not impossible, that these seizures were connected with any deranged renal action.

There are also at the present moment three cases of *erysipelas* under my care, in which this plan of treatment has been pursued with perfect success. And here let me remark, that there is no disease in which one may dogmatize so decidedly as regards the beneficial action of alcohol as in *erysipelas*. Often, indeed, it appears to me to act as a direct antidote to the erysipelatous poison; for you will find that when administered *early* and steadily, not only does the malady yield readily, but those formations of pus (secondary abscesses), which under other plans of treatment are of so frequent occurrence in this disease, hardly ever make their appearance.

The early administration of alcohol in the manner I have recommended exercises a most remarkable and unequivocal influence in preventing or materially limiting the intensity of *delirium*. Delirium is a symptom of enfeebled and contaminated nutrition of the brain. It is to be looked for in all exhausting diseases, and in all acute maladies accompanied by high fever. You meet with it in the acute internal diseases, in the exanthemata, in erysipelas, in typhus and typhoid, in influenza, in the rheumatic and gouty fevers, and after severe burns, or compound fractures, or great surgical operations, after parturition, and in profuse hemorrhage, from whatever cause. In all such cases, the timely administration of alcohol will *prevent* or mitigate the delirium, and will check it if it have come on early. It is also applicable to the treatment of delirium of the hysterical and epileptic types, although in such cases it will not in general be found to tell with such marked effect as in the delirium which accompanies acute disease.

The following is an interesting example of the latter form of delirium, occurring in connection with one of the exanthemata, and of the good effects of alcohol, as well as of the evil of deferring the administration of a proper amount of support until an advanced period of the disease.

CASE LXXXIX. A middle-aged, pale, thin gentleman, a lawyer by profession, had an attack of small-pox, by no means severe, and without copious eruption, which was not confluent anywhere. He was treated by sudorifics and aperients, and kept low on slops without broth. Just before the complete maturation of the pustules, he became violently delirious. In his excitement one day, he jumped from his bed and made for the door, and on his way his foot slipped, he fell heavily and dislocated his shoulder.

The delirium was not improved by this accident, nor by a subsequent attempt, almost immediately after, to reduce the luxation. He struggled violently and successfully against the surgeons, and, in his efforts, contrived to push himself close to a window, at which he aimed a violent kick, thrusting his foot and leg through the pane of glass, and inflicting a severe cut on the leg, which bled very freely. Soon after this, I was requested to see him in consultation with one of his medical attendants. Perceiving at once the nature of the violent delirium in which I found him, I requested that, while we were holding some conversation as to his previous history, he might have two glasses of port wine given him. This seemed to have a somewhat tranquillizing effect, and when we came to examine him, we found him more rational, and less violent. It was agreed to give him port wine very freely; at first about two ounces every hour, and a little opium and carbonate of ammonia. In the course of a few hours he began to sleep; still we did not allow him to sleep continuously for more than two hours, but had him roused to take his port wine. When the delirious state was very much subdued, he was allowed to sleep longer. Solid food was given, and the port wine was gradually and cautiously diminished. In three or four days all trace of delirium had disappeared.

Now, in this case at first there was no indication of any great depression of vital power. The absence of adequate support, and the existence of considerable febrile disturbance, gave rise to the condition which favored delirium. Had the supply been in the first instance better adjusted to the waste, delirium would not have occurred, and we should not have found it necessary to give alcohol so freely as was subsequently done.

Let me add one or two more examples to illustrate the application of this treatment to delirium in acute disease.

CASE XC. (Vol. lvii, B. p. 105.) J. Parkinson, æt. 38, a joiner, was in good health, and at work until July 30th, 1858, when he was attacked with sore throat, shivering, and headache. The throat became rapidly worse, so that he had great difficulty both in swallowing and breathing. A blister was applied over the larynx. About the fourth day he began to wander in his mind, and got out of a window on to the roof of his house. His wife stated that, four years previously, he had had a very similar, but less severe attack.

He was admitted into King's College Hospital, August 3d, 1858, the fifth day of the disease. He was then slightly delirious; there was great redness and swelling of the uvula and tonsils, and a furred tongue; fine crepitation was audible at the base of each lung.

On the 4th (sixth day), the fauces were observed to be very red, and the tonsils still swollen; and this condition was accompanied with great difficulty of swallowing. The patient appeared also to have some difficulty in speaking; every effort to do so was accompanied by a peculiar tremor of the lips and tongue preceding articulation. There was dulness on percussion, with some bronchial breathing, and a considerable amount of fine crepitation at the base of each lung behind, but rather more on the left than the right side. Pulse, 80; respirations, 18. Heart intermitting at every fifth or sixth pulsation. Tongue, brown and furred in the centre; urine albuminous; the precipitate occupying about one-fifth of the depth of the fluid. Half a drachm of the aromatic spirit of ammonia, in camphor mixture, was ordered every four hours, half an ounce of brandy every hour, and turpentine stupes to the chest.

The next day, August 5th (seventh day of the disease), the patient was rather more delirious. The fauces were still red, with a quantity of dirty, viscid mucus hanging about them. No cough or expectoration. Pulse, 84; respirations, 26. The dulness remained at the base of the left lung, but had disappeared from the right side, and there was much less crepitation. The urine contained only a trace of albumen.

At night he became more restless and delirious, and was with difficulty kept in bed. A castor oil enema was administered, as the bowels had not been moved since his admission, and the brandy was increased to half an ounce every half hour.

On the 6th (eighth day), the patient was sweating profusely. The throat was better, the fauces not so red, and the tongue cleaner. There was still some bronchial breathing at the base of either lung behind, and slight dulness on percussion remained on the left side, but crepitation had almost disappeared. The urine was free from albumen. Pulse, 78; respirations, 28.

On the 7th, there was still some delirium. Bronchial breathing had entirely disappeared. Pulse, 72; respirations, 26. Tongue clean. The brandy was reduced to half an ounce every hour; and, as the patient's bowels had not been moved, a draught of senna and aloes was given.

On the 8th (tenth day), an erythematous blush was observed, for the first time, on the left leg, extending between the knee and the ankle. Pulse, 75; heart still intermitting. The brandy was reduced to half an ounce every second hour. Two calomel and colocynth pills, to be followed by a black draught, were ordered.

On the 9th (eleventh day), the patient continued better and quite free from delirium. He was expectorating a little frothy mucus. The leg was still red and tender.

By the 10th (twelfth day), the erythematous blush had faded considerably.

On the 15th (seventeenth day), he was sufficiently well to be discharged.

Case XCI.[1] (Vol. lvii, B. p. 57.) Jane Brabbs, æt. 42, a woman of intemperate habits, after drinking hard for two days without taking food, found herself trembling, unable to sleep, and haunted by spectral illusions. In this condition she was brought to the hospital, June 11th, 1858. She was then talkative and restless, starting up frequently in bed. A drachm of tincture of opium and two ounces of brandy were administered about 4 o'clock in the afternoon, and repeated at nine; and about half an ounce of brandy was administered every hour through the night (with beef tea), but no sleep was procured.

The following day, June 12th (third of the delirium), there was less tremor, and the patient spoke rationally although rapidly. A mixture of chloric ether and ammonia with fifteen minims of tincture of opium, was ordered every four hours. In the afternoon chloroform was administered by inhalation but failed to induce sleep.

Throughout the night, brandy was given in doses of half an ounce every half hour, but was again reduced to half an ounce every hour on the morning of the 13th. The patient was then very talkative; but in the afternoon, after eating a chop, she went to sleep, and slept for three hours. She awoke better, but not free from hallucinations, and again passed a sleepless night.

During the 14th (fifth of the delirium), the patient remained awake, although the pupils were contracted and the opium continued. At night, she dozed, waking every hour for brandy. The next day, the 15th, she was much less talkative and had a fair appetite.

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[1] Reported by Dr. E. S. Thompson.

On the 16th (seventh day), as no sound sleep had been obtained, the dose of opium was increased to twenty minims, and in the afternoon chloroform was administered by inhalation. In the evening, she became noisy and violent, and the chloroform inhalation was repeated; but when its immediate effect had passed off, she was so violent that it was found necessary to remove her to a ward by herself, and to put her under restraint.

On the 17th (eighth day), the patient was still in the same excited state, violent and vociferous; large doses of opium were administered at short intervals without any benefit. At two o'clock in the afternoon, half a drachm of chloroform was given by the mouth, and shortly afterwards the dose was repeated. But it was not until a third and double dose had been given that any sleep was obtained: she then slept from six to half-past eight.

At 10 o'clock that evening, the brandy was again increased to half an ounce every hour, and the chloroform repeated. The patient slept from eleven o'clock to half-past five the following morning, the 18th, and then awoke much more rational, although still somewhat excited and trembling. The dose of chloroform was repeated.

At 12 o'clock that day, as she continued better, the brandy was reduced to half an ounce every hour, and the chloroform was ordered to be given in half-drachm doses every four hours.

Under this treatment, our patient continued to improve. On the 19th (tenth day), the chloroform was reduced to half a drachm every six hours. She slept for a good part of the night, and awoke more herself on the morning of the 20th. The brandy was then reduced to half an ounce every second hour.

On the 21st (twelfth day), although very weak and restless, she had a better appetite and less tremor, and was able to sleep for several hours consecutively.

On the 23d, she still remained very weak and slept but indifferently. The brandy was changed for a pint of porter.

On the 24th (fifteenth day), she seemed to be gaining strength, and now slept soundly. The chloroform was discontinued, as it appeared to make her sick. On the 28th (nineteenth day), she was discharged well.

The following is a case of epileptic delirium:—

**Case** XCII. (Vol. lvi, p. 202.) Caroline Lewry, aged 29, single, was admitted into King's College Hospital, Feb. 25, 1858, in a state of violent delirium.

We learned that she had been in poor health, and subject to epileptic fits from childhood; that these had recurred at intervals of about a month, always leaving her weak and exhausted for a day or two afterwards. Her mother stated that she had lately been living very badly, and that poor living always increased the frequency of the fits.

On February the 21st she had a fit, from which she recovered as usual, but on the 24th had a second attack, and from that time became delirious, and at length so violent that she was brought to the hospital.

When admitted (second day of the delirium) she had a wild expression of face, and could with great difficulty be kept in bed; she seemed to be haunted with spectral illusions, and was constantly singing. The skin was moist and cool; the tongue dry and furred. Pulse 120, weak and compressible.

Two ounces of brandy were immediately given, and half an ounce ordered every hour.

The next day, February 26th (third of the delirium) the patient remained in very much the same condition, frequently starting from her bed, and fixing her eyes on some imaginary object; but she was less noisy and violent. The pulse had fallen to 96. The brandy was continued, and a mixture containing carbonate of ammonia, with eight minims of tincture of opium in each dose, was ordered every four hours.

On the 28th (fifth day), the delirium was still of the same character; the pulse had fallen to 80.

On the 1st of March (sixth day), the patient was better—no longer violent, and able to sleep a little. Pulse 76.

On the 2d (seventh day), the delirium seemed to be passing off, and she only started occasionally and complained of a mist before her eyes and slight headache. Pulse 76. Tongue clean.

By the 3d (eighth day), our patient was quite free from delirium, and slept well. She complained of some headache and a pain in the præcordia. The heart-sounds were natural and the urine healthy. Pulse 76. Brandy reduced to half an ounce every second hour.

On the 4th, a quinine mixture was ordered. She continued improving under a tonic treatment until the 18th (eighteenth day). The headache had then ceased; she slept well, and had a good appetite, and was shortly afterwards discharged.

Let me invite your attention to the tabular statement showing the gradual decline in the frequency of the pulse under the free administration of alcohol.

TABLE OF PULSE AND RESPIRATIONS.

| | Date. | Pulse. | Resp. | |
|---|---|---|---|---|
| Brandy ℥ij immediately, and ℥ss every hour afterwards. | Feb. 25th | 120 | | During the delirium. |
| | " 26th | 96 | | |
| | " 28th | 80 | 24 | |
| | March 1st | 76 | 24 | |
| | " 2d | 76 | 26 | |
| | " 3d | 76 | 26 | |
| | " 4th | 78 | 30 | Delirium ceased. |
| Brandy ℥ss every 2d hour. | " 6th | 80 | 30 | |
| | " 9th | 78 | 28 | |
| | " 11th | 80 | | |
| | " 13th | 80 | | |

Case XCIII.[1] (Vol. xlix, p. 75.) Harriet Smith, aged 38, while residing in the same house with a person ill of fever, began to feel unwell, and was soon afterwards attacked with an intense headache. She quickly lost all consciousness, and now that she is well, remembers no further particulars of her illness.

This patient was brought to the hospital, July 29th, 1855, about a week from the commencement of the malady, in a state of almost complete insensibility. When put to bed, she lay nearly motionless, picking, however, at the bed-clothes with her fingers, and occasionally moaning or muttering, or crying out if moved. When loudly spoken to, she appeared to be somewhat roused, but not sufficiently to answer questions. Both urine and fæces were passed unconsciously in the bed. A number of fawn-colored spots were observed scattered over her body, especially on the chest and abdomen. Pulse, 135.

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[1] Reported by Mr. J. H. Thornton.

A mixture of chloric ether and ammonia was ordered every third hour, an enema of ten grains of quina, in two ounces of beef tea, three times in the day, and half an ounce of brandy, with beef tea, every hour.

On the 30th, the pulse and respirations were 115 and 28; on the 31st, they were 116 and 24. The patient remained in the same state of stupor, picking and muttering, but taking her food and brandy well. She was also perspiring freely. No diarrhœa nor relaxed motions.

On August 1st, about the 11th day of the disease, she was rather more sensible; skin hot, but perspiring less; pulse, 120; respirations, 28.

On the 2d, the pulse and respirations were 114 and 24 respectively; on the 3d, 108 and 24. The patient was better, and had left off muttering, and picking the clothes, but now began to vomit her food and medicine.

On the 4th (fourteenth of the disease), she was much better, and able to give rational answers to questions, but still passed her evacuations unconsciously, and the vomiting continued. Tongue, cleaner; pulse, 120; respirations, 20.

On the 6th (sixteenth day), the pulse and respirations were 106 and 20; on the 8th, 100 and 20. Tongue much cleaner. The patient was still troubled with sickness.

On the ninth, a quinine mixture was substituted for the chloric ether and ammonia, and the following day the sickness entirely ceased. The patient now complained of pain in one knee. Pulse, 96; respirations, 24.

On the 13th (twenty-third day), she was still suffering great pain in her knee, and the pulse had risen slightly to 108.

By the 16th (twenty-sixth day), there was a very great improvement. Our patient was fairly convalescent, and able to eat and sleep well, and to get up for a short time. Pulse, 84. The brandy had been gradually reduced. She continued to improve, and gain strength, until the 25th, when she was discharged well.[1]

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[1] Other examples of recovery from a comatose state in fever, under large doses of alcohol, will be found at pages 120 and 97.

You will ask, what are the signs that too much alcohol is being given, and what are the indications which should suggest to the practitioner that he ought to diminish the quantity, or to withdraw it altogether.

I must tell you, *in limine*, that it is far more dangerous to life to diminish or withdraw alcohol than to give too much. It is plain to common sense that, in the latter case, you may easily and quickly reduce the quantity, or suspend the use of alcohol altogether; but it is not so easy to make up for lost time, and to supply readily a want which had been felt for many days. Hence it sometimes happens (as in Case LXXXIX), that the withholding such a supply in the early period of acute disease, leads to the employment of a much larger quantity in the latter stages.

The signs upon which I recommend you to place your chief reliance, as indicative of the supply being too much, are mainly referrible to the digestive organs. They are such as show that the first stage of digestion—stomach digestion—is disturbed. Your patient will suffer from flatulence, frequent eructations, even sickness, dryness of the tongue and mouth, with some degree of sordes of the teeth and lips. Of course you will take pains to separate such symptoms as arising from too much alcohol, from similar ones as belonging to the disease.

It is important that you should watch the effects upon the nervous system. When alcohol is given as I have recommended, it calms the nervous system, promotes tranquil sleep, from which the patient may be easily aroused, and *averts* delirium. If given in excess, either by large doses at once, or by too frequent doses, it tends to produce coma. In the first case, the quantity is disposed of partly by being appropriated to the nutrition of the nerve matter, whether in centres or nerves, and partly by combustion and elimination, as carbonic acid and water. In the other case, there being more than sufficient for the normal wants of the system, a large proportion remains in the blood as alcohol, or passes into the more simple secretions, such as the fluid of the cerebral ventricles, and acts as a poison on the nerve-fibre and nerve-cell, disturbing their nutrition, so as to produce the phenomena of drunken or alcoholic delirium, or paralyzing their nutrition, and inducing coma.

The coma and the delirium produced by an excessive use of alcohol are often difficult to distinguish from these phenomena, as forming part of an acute morbid process, and as indicating rather the need of a more active exhibition of alcohol. Although over-stimulation is of very rare occurrence in our practice, you will do well to be provided with some signs to distinguish it when it occurs. The following points are, I think, deserving of your attention :—

1. The coma of alcohol is not so profound as that of disease; and the patient who is suffering from the former may be more easily roused than in the coma from disease. It is a good plan in these cases, as a test between the two forms of coma, to sluice the head well with cold water. The patient with coma of alcohol will much more readily respond to this than when the comatose state is part of the disease.

2. By omitting the alcohol for two or more hours, and giving nothing but water for that time, and visiting your patient after the lapse of a short period, you will find the coma distinctly less if it have been excited by alcohol. But in such an experiment as this, you must watch the patient at short intervals, and be very cautious of withholding the stimulant for too long a time. I have often known patients sink irretrievably, through timid practice, when the alcohol had been too long withheld.

3. The persistence of the exhalation of the fumes of alcohol by the breath, may be regarded as a sign that too much is being administered. When the just quantity is given, the fumes are not perceived in the breath, or at most for but a short time after a dose has been taken.

I shall conclude this already too long lecture with a few additional remarks on the case upon which it is founded.

It is quite clear that all was done in this case that ought to have been done to restore the child; that nothing was omitted—nothing done too much.

The case was complicated a good deal by the vomiting which occurred towards the close of it. I do not see, that at the early period of the symptoms more could have been done than was done. At that time, the nerve-fibre and nerve-cell were paralyzed by the overdose of alcohol, and it was necessary to wait until the spirit had been consumed or eliminated. After that, there still remained a comatose state, from the want of the renewal of the ordinary nutrition. We then ventured to give small quantities of wine. Were I to treat such a case again, I would give wine or brandy more freely than was done in this instance, and I would also administer by the rectum, quinine with a small quantity of brandy. You will find this often a very useful practice where the powers of life are low, and it ought not to have been omitted in our little patient's case. But she showed so little power of reaction, that it is in the highest degree improbable that any further treatment would have been successful.

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